‘’A STUDY OF PROFILE,MANAGEMENT AND OUTCOME OF PATIENTS ADMITTED FOR SNAKE BITE WITH ENVENOMATION IN GENERAL MEDICINE DEPARTMENT, GOVERNMENT CHENGALPATTU MEDICAL COLLEGE & HOSPITAL”

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DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation titled "**A STUDY OF PROFILE, MANAGEMENT AND OUTCOME OF PATIENTS ADMITTED FOR SNAKE BITE WITH ENVENOMATION IN GENERAL MEDICINE DEPARTMENT, GOVERNMENT CHENGALPATTU MEDICAL COLLEGE & HOSPITAL**"

is a bonafide and genuine research work carried out by me at the **GOVERNMENT CHENGALPATTU MEDICAL COLLEGE & HOSPITAL** from June 2013 to May 2014 under the guidance and supervision of **Dr.R.MUTHUSELVAN**..M.D, Professor , Department of General Medicine, GOVERNMENT CHENGALPATTU MEDICAL COLLEGE & HOSPITAL Chengalpattu-603001

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CERTIFICATE

This is to certify that the dissertation titled "**A STUDY OF MANAGEMENT AND OUTCOME OF PATIENTS ADMITTED FOR SNAKE BITE WITH ENVENOMATION IN GENERAL MEDICINE DEPARTMENT, GOVERNMENT CHENGALPATTU MEDICAL COLLEGE & HOSPITAL**" is a bonafide work of

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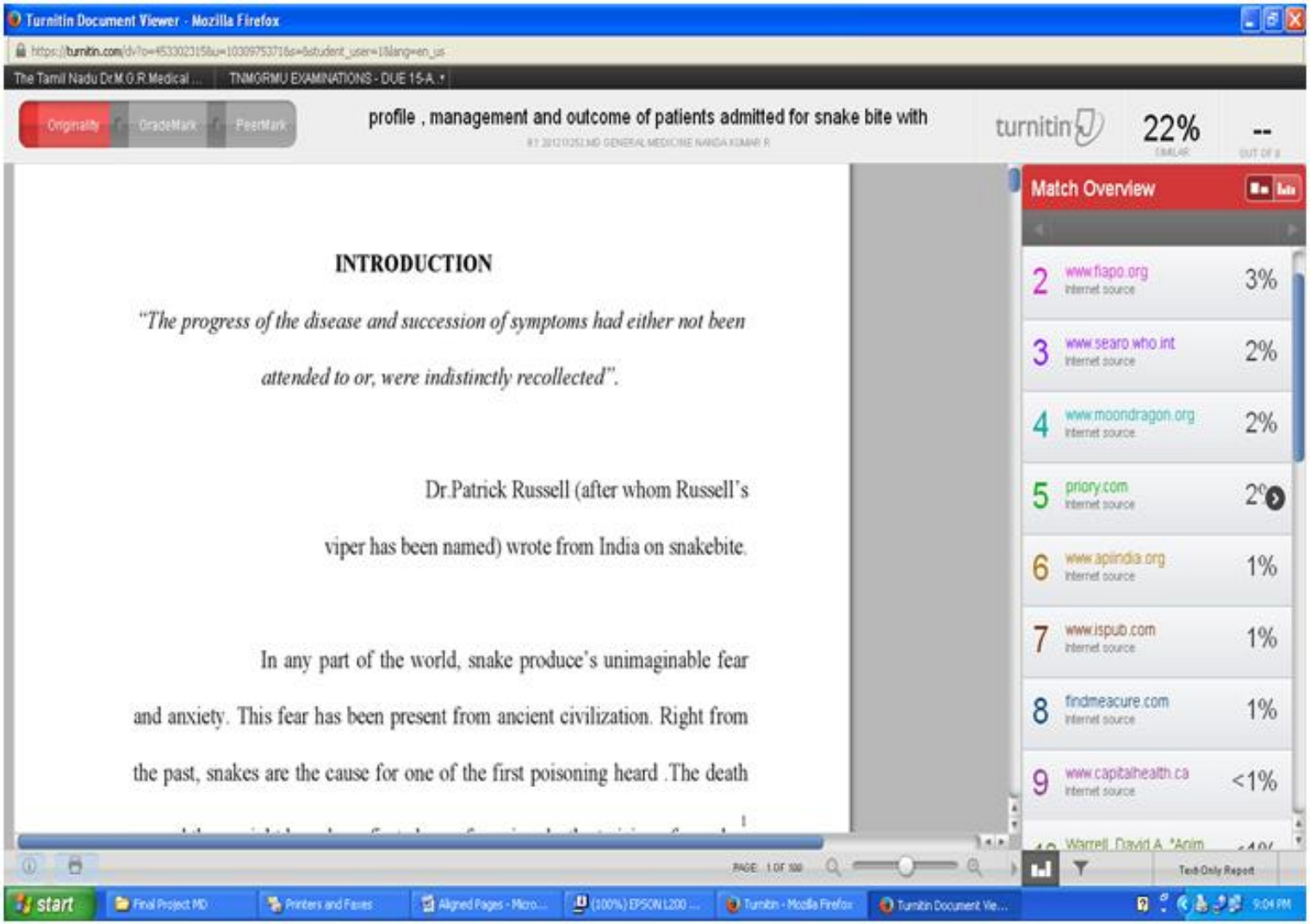

INTRODUCTION

"The progress of the disease and succession of symptoms had either not been attended to or, were indistinctly recollected".

Dr.Patrick Russell (after whom Russell's viper has been named) wrote from India on snakebite.

In any part of the world, snake produce's unimaginable fear and anxiety. This fear has been present from ancient civilization. Right from the past, snakes are the cause for one of the first poisoning heard .The death

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I bow my head in respect before God Almighty.

Date: **Signature of the Candidate**

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LIST OF ABBREVIATIONS USED

A.....Absent

AMA.....Against Medical Advice

ASV.....Anti snake Venom

BTN.....Bite To Needle Time

BA.....Bronchial Asthma

CAHD.....Coronary Artery Heart Disease

DC.....Differential Count

DM.....Diabetes Melitus

E.....Expired

ECG.....Electrocardiogram

F.....Female

H.....Haematotoxic

I.....Impaired

M.....Male

N.....Neurotoxic

O.....Observed

P.....Present

RV.....Russell Viper

SSV.....Saw Scaled Viper

TC.....Total count

WBC.....Whole blood clotting

WHO.....World Health Organisation

WNL.....Within normal limit

Yrs.....Years

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ABSTRACT OF THE STUDY

TITLE :

A STUDY OF PROFILE,MANAGEMENT AND OUTCOME OF PATIENTS ADMITTED FOR SNAKE BITE WITH ENVENOMATION IN GOVERNMENT CHENGALPATTU MEDICAL COLLEGE & HOSPITAL,CHENGALPATTU .

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KEYWORDS :

Snake bite envenomation, Bite to needle time ,Co morbid illness, native treatment, Tourniquet application, Anti snake venom , Fasciotomy.

ABSTRACT

BACK GROUND AND OBJECTIVES :

Snake bite is a major public health problem through out the word especially in tropical countries like India. The objective of the study is to analyse the factors, which determine the prognosis of patients, admitted with snake bite envenomation . These prognostic factors include 1) Time since snake bite and admission to Government Chengalpattu Medical college & Hospital, Chengalpattu 2) Whether patient received native treatments before admission . 3) Co morbid illness. 4) Site of bite.

METHOD:

181 adult patients admitted with signs of snake bite envenomation were included in this study and a detail history of bite including time of bite, site of bite, type of snake, patients manifestation and history of native treatment received before admission was taken .A detailed history of co morbid illness was obtained from all patients. A detailed clinical examination was done and each patient was subjected to investigations like complete blood count, BT, CT, PT, blood urea, serum creatinine, electrolytes and ECG. All investigations were repeated to evaluate the progress

RESULTS :

Total cases studied are 181, in which 64% are males and 36% are females .Highest incidence of snake bite was seen in **1**. Age group of 30-40 yrs(26.8%) **2**. Purely agriculture workers (46.41%) .There was more incidence of bite on lower extremity (50.83%). Complications are low in patients presenting within 6 hours of bite. Out of 181 cases of snake bite 35.38%were neurotoxic, 25.42% were haemotoxic and Local reactions like cellulitis , edema comprises 22.65 % and of these 9 patients expired .

INTERPRETATION AND CONCLUSION

1) A significant association was noted between prolonged bite to needle time and mortality with 7 out of 9 patients(78%) who presented more than 6 hours after bite were dead .
2) There was no relation between site of bite and severity of envenomation

3) On analysing co morbid illness versus death wise distribution of study population , we found that among 15 diabetic patients 93 % were alive and 7% were dead . Among 17 hypertensive patients only 6% died . Similarly on analysing all the co morbid illness with outcome we found that co morbid illness is not a strong determinant in determining the mortality.

4) Native treatment especially tourniquet application is a strong determinant of outcome with 100% prevalence in dead and 85.28% in patients who had undergone surgeries and 27.28 % in alive patients without surgeries .

INTRODUCTION

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Dr.Patrick Russell (after whom Russell's viper has been named) wrote from India on snakebite.

In any part of the world, snake produce's unimaginable fear and anxiety. This fear has been present from ancient civilization. Right from the past, snakes are the cause for one of the first poisoning heard .The death caused then, might have been first alarm of sensing death at vision of a snake[1]

At the beginning of twenty first century, annual mortality from snakebites continues to be as high, around 30 to 60 thousand in the world. Snakebite morbidity and mortality is a major health problem in rural areas. Mortality rates of around 5.5 per 100,000 resident Indian rural coastal populations tell about the magnitude of the problem. According to Frayer in his study of Thanatophidia of India,it was estimated that about 1 in 1 lakh population died due to snake bite[2] .But inspite of this, snake bite has been overlooked through ages.

Snake bite is also responsible for about 2.85%-5.3% of the mortality of total hospital admission in India. India is reported to have the highest snakebite incidence and mortality in the world[3]. World Health Organization (WHO) estimates the total number of bites to be 84,000 per year with 11,000 deaths. A national survey in India suggests that snake bite deaths estimated are more than 30 fold higher than documented. Most of the death are mainly due to the snake bite victims not reaching the hospital in time.

The increased mortality in India is due to climatic factors, rural predominant population and their agricultural dependence. In addition high risk community is not well educated , about the occupational risks of exposure and simple precautions like wearing slippers which can protect against the bite. Also many harmful practices such as cutting and suction, tourniquets application , sucking out venom with mouth, etc are followed in many places which all contribute to mortality and morbidity due to snake bite.

Increased accidental exposure to snakes, due to agricultural practices, lack of good health care services, poor access to tertiary care services and lack of effective anti venom all contribute to the morbidity and mortality .

Studies signify that primary care treating doctors hesitate to treat snakebite patients immediately mainly due to lack of experience and confidence. At the secondary and tertiary level hospitals, several treatment protocols and schedules were being followed for anti-snake venom (ASV) administration, mainly based on foreign textbooks. Mortality rate is further increased by inappropriate administration of first dose of ASV particularly in krait and Russell's viper snakebite[5]. There is also delay in providing a simple method of endo tracheal intubations and artificial ventilator or amboo bag ventilation in neuro-toxic envenoming.

In response our Health and Family Welfare Department, Government of India, after careful analysis of trials has prepared a National Snakebite management Protocol for snake bite to provide doctors and lay people a best evidence-based treatment approach to deal with this problem in our country[6].

Around 235 species of snakes are found in India, most of which are non venomous. Most of the bites , will cause panic reaction , but do not cause envenomation . However, there are few snake species that are venomous and of these four(Big four), namely Russell's viper (*Dabiola russelii*), common cobra (*Naja naja*), saw-scaled viper (*Echis carinatus*) and

common krait (*Bungarus caeruleus*) are highly venomous and believed to be responsible for most of the poisonous bites in India[7].

In our Government Chengalpattu Medical college & Hospital, situated in kanchipuram district , snake bite cases comprises a major proportion among hospital admissions. We are well equipped and trained in managing this most common problem. In spite of good care and timely management some cases end up in complications or death .

Main reasons are, patients presenting lately for treatment due to lack of awareness , patients undergone native treatment and then getting admitted to hospital with complications , co morbid illness of the patients. In 2012 total number of snake bite cases were 424 out of which 167 cases were poisonous bites with signs of envenomation .

In context of the above facts this study entitled "*A STUDY OF PROFILE,MANAGEMENT AND OUTCOME OF PATIENTS ADMITTED FOR SNAKE BITE WITH ENVENOMATION IN GOVERNMENT CHENGALPATTU MEDICAL COLLEGE & HOSPITAL,CHENGALPATTU GOVERNMENT HOSPITAL''* was undertaken to study the prevalence and the role of different factors in determining the outcome in snake bite envenomation patients .

AIMS AND OBJECTIVE

This study was carried out from June 2013 to May 2014 (one year Study), the study was conducted prospectively . The study comprised of 181 cases of snakebite patients(>13 years) with signs of envenomation admitted to the Department of General Medicine , Government Chengalpattu Medical college & Hospital ,Chengalpattu.

1. To know the age and sex distribution of snake bite poisoning .
2. To study the high risk group involved and things that can be done to prevent the incidence among them .
3. To study distribution of cases based on time, place and site of bite .
4. To study the toxicity, complications, treatment and outcome of snake bite envenomation in adults.
5. To study the effect of factors like Bite to Needle time, native treatment and co morbid illness in determining the outcome.

REVIEW OF LITERATURE

The term ‘Snake’ was derived from the Anglo-Saxon word ‘Snaca’ meaning the creeping[8]. Nearly, 240 million years ago these creeping animals were seen and were named ‘reptiles’.

Dr.Patrick Russell[9] is the “Father of Indian Ophiology” .He gave the earliest reference to Indian Snakes. He holds the credit for distinguishing the venomous from the non-venomous snakes. It was he who focused attention on the viper Vipera Russelli, which was named after him.

Sir J.B.Fayrer[10] (1873) was a physician and ophiologist .He has done extensive research on the physiology of venom of Indian snakes. He wrote a book in 1874, named “Thanto Ophidia of India”.

A book on the poisonous snakes of India ,by Joseph Ewarts[11] (1878) provides valuable datas on snake bite poisoning in India.

An, ophiologist named, Col. Frank Walls[12] (1908) strived hard to gain insight on knowledge of the habits and distribution of Indian snakes. His work entitled “The poisonous terrestrial snakes of our British Indian Dominions and how to recognize them “bears his stamp of authority on the subject.

Col. Gharpurey[13] (1935) a physician, first attempted to dispel the superstition and ignorance woven around the Indian snakes, both venomous and non-venomous book entitled “Snakes of India “which was written in semi-technical language by him, provides plenty of information information on the venomous snakes.

Dr.Malcolm Smith[14] (1943) ,volume on ‘’snakes in the Fauna of British’’ Indian series continues to be a gold standard work. Information on the clinical aspects of snake bites in India and Southeast Asia has been given by Alistair Reid H[15] (1963) .Similarly Banerjee RN and Siddiqui ZA[16] (1974) ,Of Safdarjung Hospital, New Delhi conducted a study of snake bites, which includes many aspects of envenomation. Sawai et al[17] of Japan Snake Institute, published a statistical data on snakebite based on records of the patients from different states in India ,during his visit to India from October to December 1972.

Several medical personals have conducted studies and investigations on the venoms of snakes in India and their properties . The list of such workers is very long and exhaustive, some pioneering work carried out in recent times by Ahuja and Gurkripal Singh[18] (1954), Deoraj[19] (1963-1971) and Whitaker[20] (1978), Daniel[21] (1983) has given details facts and figures in respect of the venom of the common venomous snakes.

Murthy[22] (1985) studied about the common venomous snakes of India and also gave valuable accounts of these snakes for the benefit for layman. Three famous institutes in India contributed to various research on snake bite namely, The Haffkine Institute for Training, Research & Testing in Mumbai, The Guindy Snake Park, The Snake Park in Calcutta and the Central Research Institute, Kasauli .They served as a source for the dissemination of knowledge ,about venomous snakes of India and their venom, preparation of antisnake venom sera and the treatment of snake bite, etc.

Acoording to studies conducted by Subhadeep Sarkar, Parthsaratho Mitra and Kunal Bhattacharya , Russel's viper is responsible for 60 percent of the deaths from snakebite, that occur in India every year. Death due to snake bite is due to harmful effect of all the toxic reactions that venoms produce in the body of the victim.

Snake venom is just a modified saliva with a combination of many different toxic proteins and enzymes. These constituents exert toxic effects on entering the biological systems. Inside the snake ,venom is present outside the circulation and physiological compartment of body, confined to venom gland and so snakes are likely to suffer from toxicity of venom, when bitten by other venomous snakes. A highly potent natural antivenom substance with greater potency and prolonged half life has been demonstrated in rattle snake (Straight et al, 1976).

CLASSIFICATION:

All snakes in India commonly fall basically into two families[41].

1. Under Family colubridae, there are three sub-families.
   a) *Elapidae*:,These include most of the neurotoxic snakes like Krait, Coral snakes, Cobra and Mamba .
   b) *Crotalidae*:it comprises pit viper and rattle snakes.
   c) *Hydrophidae*: Sea snakes.

2. Family Viperidae (True Vipers): These comprises many African and Eastern burrowing Stiletto snakes, also known as burrowing vipers or mole vipers .Usually attack their victims with front fang protruding through the partially closed mouth.

The main characteristics of important poisonous snakes of India are:

a) Elapids:

This group comprises of cobra, krait and the coral. The head and neck are of the same width and the pupils are round. The fangs are located anteriorly but being covered by mucous membrane. Therefore these snakes cannot bite through the dressings or inject a completely. The tails are rounded.

1. Indian Cobra:

The snake measures up to 2 meters. Its colour is variable but usually black. The head and neck are of same width, which is provided with a hood, bearing a mark. It spreads its neck to form a hood only when frightened. The cobra prefers populated areas and it has very vast distribution in India.

FIGURE 1: INDIAN COBRA

2. King Cobra:

This snake is larger and bigger than common cobra, it can grow up to 4.5 meters. The colour is variable from jet black to yellow, brown or black.

It has a hood without a mark . The tail scales are arranged proximal but with distal division. The king cobra resides in dense jungles or forests.

FIGURE 2 : KING COBRA

3. COMMON KRAIT :

This snake grows up to 1.50 meters. It is usually glistening black colour and has white arches across the back beginning from the head. It has a row of hexagonal central scales on the back with a creamy white belly. Large shields cover the head with complete tail scales. Usually prefers to reside near the house and is responsible for a large number of cases of poisonous snake bite in India.

FIGURE 3: COMMON KRAIT

4. Banded Krait :

This snake is much bigger than the common krait and grows up to 2 meters. In addition to the features of the common krait, as the name implies it has alternate yellow and black bands in its back. It is commonly found in Assam, Bengal and many regions of south India.

FIGURE 4 : BANDED KRAIT

Vipers:

This group is classified as pit vipers and pit less vipers. The pit which is situated between the nostril and the eyes help them to detect prey in the dark. The shape of head is triangular much wider than the neck and the pupils are vertical. They have a long fang that is movable and canalized like a needle. Thus this snake can bite through dress and gives a full dose.

The fangs are clearly visible when erected but it is tucked by the side of the upper jaw. The bites of pit vipers are not fatal while those of pit less vipers are harmful to man. The tail is tapering. The bamboo snakes are pit vipers while the Russell's viper and saw-scaled viper are pit less vipers.

1. **Russell's Viper** :

This snake grows up to about 1.5 meters; it is brown in colour and has three rows of black spots or chains on the back. It is stouter than all other poisonous snake, it can be identified by:

a) A flat head with a distinct 'V' mark with its forward pointing apex .

b) Small head scales

c) Broad belly plates and

d) A short tail with covering shields . It has bigger nostrils than other snakes. It makes a distinct hissing sound, at time of bite. It is widely found in dense jungles.

FIGURE 5 : RUSSELL'S VIPER

2. Saw Scaled Viper (*Echis Carinata, Phoorsa, Echis* or *Afai*) (Figure-9):

This snake grows up to 70 centimetres. Its is brown or brownish grey in colour. It shows following features.

a) Head resembles a triangular arrow with a white mark an arrow.

b) A diamond shaped area on each flank between the curves of wavy lines.

c) Small scales in head.

d) Broad belly plates

e) Undivided shields in tail and

f) Body scales appear like a saw and hence it is named so . It makes a distinct rustling sound while moving . This snake is found throughout India.

FIGURE 6 : SAW SCALED VIPER

POISONOUS VS NON POISONOUS SNAKES:

Because the most of poisonous snakes are members of the pit viper family, we can easily differentiate between poisonous and harmless snakes. The three ways to distinguish poisonous snakes:

Pupil Shape*:* Pupils of harmless snakes are round. Poisonous snakes have cat-like (elliptical) pupils. In good light, we can easily see the pupil shape from a safe distance because snakes cannot jump nor can they strike from more than one-third of their body length.

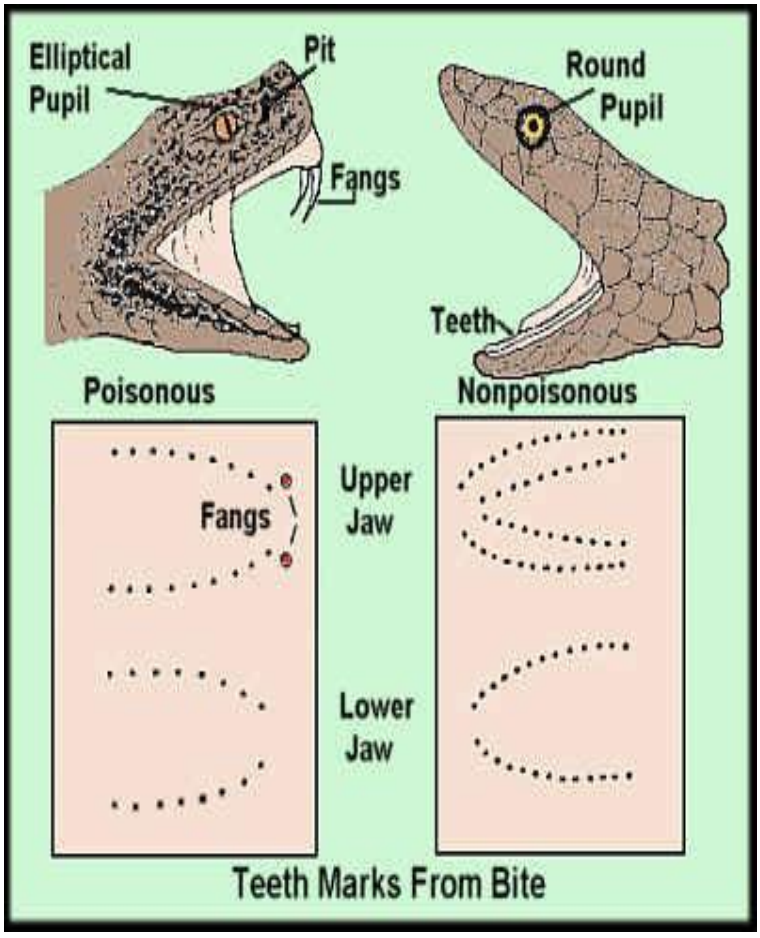
Pit: Poisonous snakes also have a very conspicuous sensory area or pit (hence the name "pit viper") on each side of the head. The pit looks somewhat like a nostril and helps the snake locate warm-bodied food. It is located about midway and slightly below the eye and nostril. Harmless snakes do not have pits.

FIGURE 7: VIPER WITH PIT

SCALE ARRANGEMENT:

The underside scales of a venomous snakes tail go all the way across in a single row from the anal plate. The very tip of the tail may have two scale rows. Non-poisonous snakes have two rows of scales from the vent to the end of the tail. This characteristic can also be observed on skins that have been shed.

FIGURE 8: POISONOUS VS NON POISONOUS FANGS



Other features that help to identify a poisonous snake at a distance:

Head Shape: All Venomous snakes usually have a triangular (wide at the back and attached to a narrow neck) or "spade-shaped" head. Sometimes many harmless snakes can also flatten their heads when threatened and may appear poisonous.

Distinctive Sound*:*

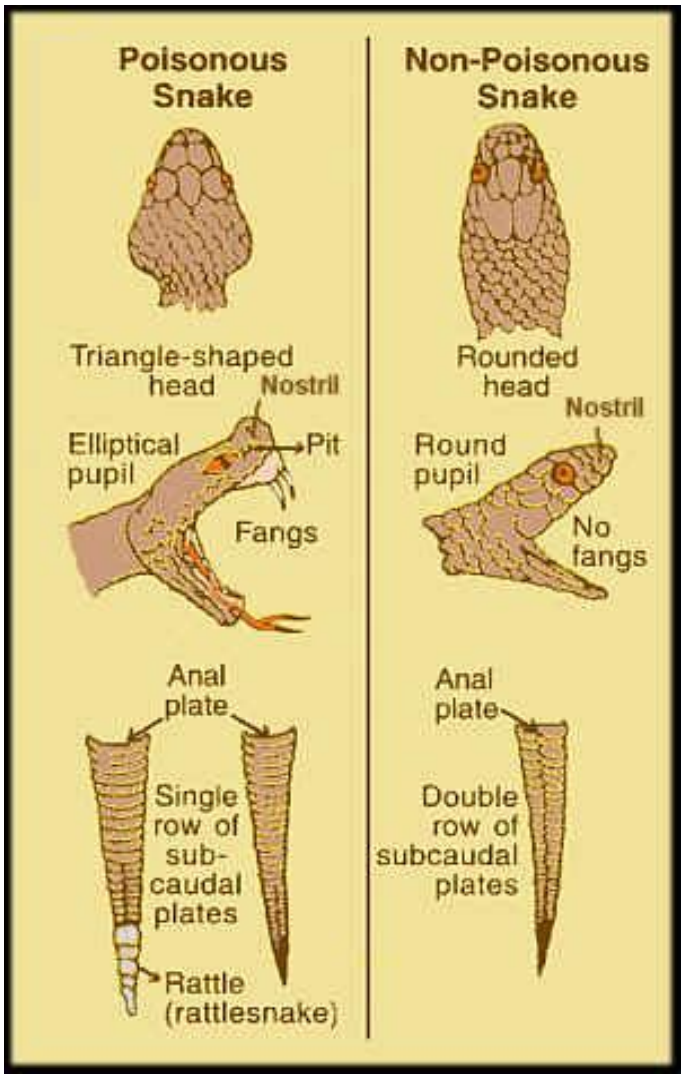
Rattlesnakes usually produce a warning rattle (a buzz or a dry, whirring sound) when approached. However, many non-poisonous snakes (black racers, rat snakes, milk snakes, and pine snakes) and several poisonous snakes (copperhead and cottonmouth) often vibrate their tails when threatened. The sound produced by this vibration often imitates a rattle or hissing sound when the snake is sitting in dry grass or leaves.

FIGURE 9 : RATTLESNAKE RATTLES

TAIL: It can be easily recognized by young cottonmouths and copperheads by their bright yellow or greenish yellow tail**.**

FIGURE 10 : POISONOUS AND NON POISONOUS SNAKE

DIFFERENCE



Site of bite and bite marks :

Most of snake bites are common during evening and night time . most common site of bite is lower limb . But other sites like hand , head and neck may also be involved . Many research works have been done in past to find the relation between site of bite and toxicity .

FIGURE 11 : SNAKE BITE IN FOOT

FIGURE 12 : SNAKE BITE WITH CELLULITIS IN HAND

Venom Composition[23,24] :

Snake venom is a mixture of several chemical compounds. Charles Lucien Bonaparte , brother of Napoleon Bonaparte, was the first to describe the proteinaceous nature of snake venom in 1843.

Proteins constitute 90-95% of venom and they are responsible for almost all of its biological effects. Most of these are enzymes which are mentioned below

1. Neurotoxins
2. Fibrinolysins

3. Cholinesterase

4. Proteolysins

5. Hemolysins

6. Agglutinins

7. Cardiotoxins

8. Coagulase, hyaluronidase, lecithinase, etc.

Inflammatory mediators such as histamine and 5 hydroxytryptamine may be partly responsible for the pain at region of snake bite

Neurotoxins (Polypeptide Toxins):

These are low molecular weight proteins found in elapids, vipers and hydrophids. They chiefly act at the peripheral neuromuscular junction ,either acting pre or post-synaptically. They either block the release of acetylcholine from the pre synaptic terminal or its interaction with its receptors, thereby preventing conduction of impulses to muscles especially those associated with breathing and extra ocular muscles. Post synaptic neurotoxins in cobra venom, such as cobratoxin and α-bungarotoxin bind to acetylcholine receptors on the motor endplate. Presynaptic neurotoxins in krait ,such as β-bungarotoxins, crotoxins are phospholipases A_2 which prevents the release of acetylcholine at neuromuscular junction by blocking voltage gated potassium channels.

Haemolysins:

Snake venom affects almost all components of hemostasis like vascular wall,platelet,coagulation,fibrinolysis [24].Haemolysins are responsible for haemorrhagic activity in viperidae venoms and it causes haemorrhage by damaging vascular endothelium and by inhibiting platelet aggregation. Procoagulant enzymes mainly acts at various points of the clotting cascade while fibrinolytic factors may act directly or by activating plasminogen. Fibrinolysis and associated platelet abnormalities causes persistent bleeding from vessels.

Myotoxins;

Produce muscle necrosis and myoglobinuria.the ongoing inflammation will cause edema and some time causes rhabdomyolysis.

Proteolytic Enzymes: causes breakdown of tissue proteins, damage cells and tissues at the site of bite causing local pain and swelling. Thus they also favour penetration of toxins to deeper tissues.

Hyaluronidase: Facilitates penetrations of venom into the tissue and its rapid absorption. It produces edema,localised swelling and absorption of the toxin at the site of bite. Other enzymes include collagenases,

phosphoesterases, monoesterases, acetyl cholinesterase, ribonucleases, deoxyribonucleases, lactate dehydrogenases. The role of these enzymes in human envenoming is uncertain.

SYMPTOMATOLOGY:

The symptoms and signs in a patient with snake bite envenomation are dependent upon a number of factors.

1. Age and General Health of the Victim:

Children and elderly persons are more likely to succumb easily because of poor general health condition .Younger patients are also at a greater risk because of higher concentration of snake venom in relation to the body volume .Old persons with features of dementia and visual disturbances are at high risk.

2. Amount of Venom Injected:

A bite by a poisonous snake may not be always poisonous.This depends on factors such as condition of the fangs and venom apparatus , such as when snake bites a man after having a recent kill of prey it will be able to inject only small quantity of venom or no venom at all. This is the reason why only some persons develop signs of envenomation after a poisonous snake bite.

Other factors like the kind of clothing at time of bite through which the fangs pass, factors tha make the snake to bite and the length of time the snake holds on will determine the amount of venom injected.

3.Nature, Location, Depth and Number of Bites:

More deeper and more the number of bites, more likely it will be lethal ,due to the direct injection of venom into a blood vessel .It will lead to rapid dissemination of venom in to circulation and severe systemic envenomation. Bite through clothing is less dangerous than bite on a bare limb. Some studies have shown that bite in upper limb and face is more likely to be lethal than in other sites

4. Type of Snake Involved:

This will decide the symptomatology. If the snake is identified as poisonous or non-poisonous and amongst the former as Elapidae (neurotoxic)or Viperidae(hemotoxic) much of the clinical signs and symptoms can be predicted and managed accordingly. Bite by a venomous snake needs urgent management instead of mere observation in case of non venomous or unknown snake bite

5. Individuals Sensitivity to Venom:

It will vary from person to person. Based on this various studies have been conducted regarding desensitization as a form of protection against snakebite. Anaphylactic shock can occur in a person repeatedly bitten by snakes.

6. Economic status :

Relationship of poverty and snakebite incidence and mortality has been clearly demonstrated[26] . The Day to day survival of many of the rural poor, in India and other developing regions of the world, like Africa are based on low cost, non mechanized farming , which puts them at risk of bite.

7. First aid treatment and Subsequent Medical Care:

This is the important factor, which is under our control . Proper first aid management and timely intervention will naturally decide the prognosis. Even in case of venomous snakebites, over half of the victims escape without serious poisoning. Snake venom poisoning may vary from trivial symptoms to extremely grave outcomes . So timely administration of ASV and early referral to tertiary centres improve the outcome.

8. Pathogens Present in the Snakes Mouth:

Apart from toxic effects of snake venom, gram negative and anaerobic organisms are infiltrated at the region of bite . If infection develops, clinical clue to anaerobic infection will be presence of a foul odour. This will add to morbidity especially in diabetic patients.

9. Absorption of the Venom:

Systemic absorption of the venom is mainly via the lymphatic route, although venous absorption can also occur with low molecular weight venom such as cobra venom. Bite over a blood vessel causes very rapid envenomation and systemic manifestations and the mortality is very high[25].

10. Native treatment :

Many people in rural areas go for native treatment for snake bite .Many scientifically disproved treatments like tourniquett application , cutting, puncturing etc, carries more harm . Many studies have shown the possibility of surgical complications and amputations in those cases .Mortality also high in such patients.

FIGURE 13: TOURNIQUETT APPLICATION

Seasonal Incidence:

There is a definite seasonal pattern in cases of snakebites poisoning. Most of the cases were reported in the 3rd quarter of year i.e., July to September. This is due to the fact that this is the monsoon season and rainfall forces snakes to venture out of their water-filled pits and there is increased human activity in fields in at this period as it is the sowing season. A similar trend was observed by Viramani SK and Dutt OP[64].

CLINICAL FEATURES OF SNAKE BITE:

The symptoms and signs of envenomation depends on the nature of the venom, the dose and the site of injection[26]. The clinical features of snake bite can be divided into local and systemic.

1. Local Manifestation:

Pain: Local pain and swelling are the commonest symptoms, it usually starts within minutes of the bite. During the next few hours pain intensifies and spreads towards the trunk and becomes localized in the lymph nodes draining the bite site. Symptoms are more rapid with cobra bite. Some

patient complains of vague abdominal pain (especially in krait bite) within 6 hours of bite.

Swelling: Swelling usually develops at the site of bite within minutes up to 48-72 hours. It will become greatest at 1 to 4 days. Both local pain and swelling is more in viper bite[27]. This is followed by oedema, and appearance of bullae, which can progress rapidly to involve the trunk[28].

Neurological symptoms:

Acute neuro muscular weakness with respiratory failure is most important manifestation. Most of the patients with neuro toxic bites develop ptosis and opthalmoplegia .Sensory disturbance like Tingling and Numbness over the tongue, mouth, scalp and around the wound occur mostly in viper bites.

Weakness of the muscles increases and develops into paralysis of the lower limbs. The paralysis then spreads to the trunk and affects the head, which droops. After half to one hour, there is excessive salivation and even vomiting. Single breath count is performed to identify the patients in impending respiratory failure and plan for intubation.

FIGURE 14 : PTOSIS IN AN YOUNG MAN WITH NEUROTOXIC SNAKE BITE

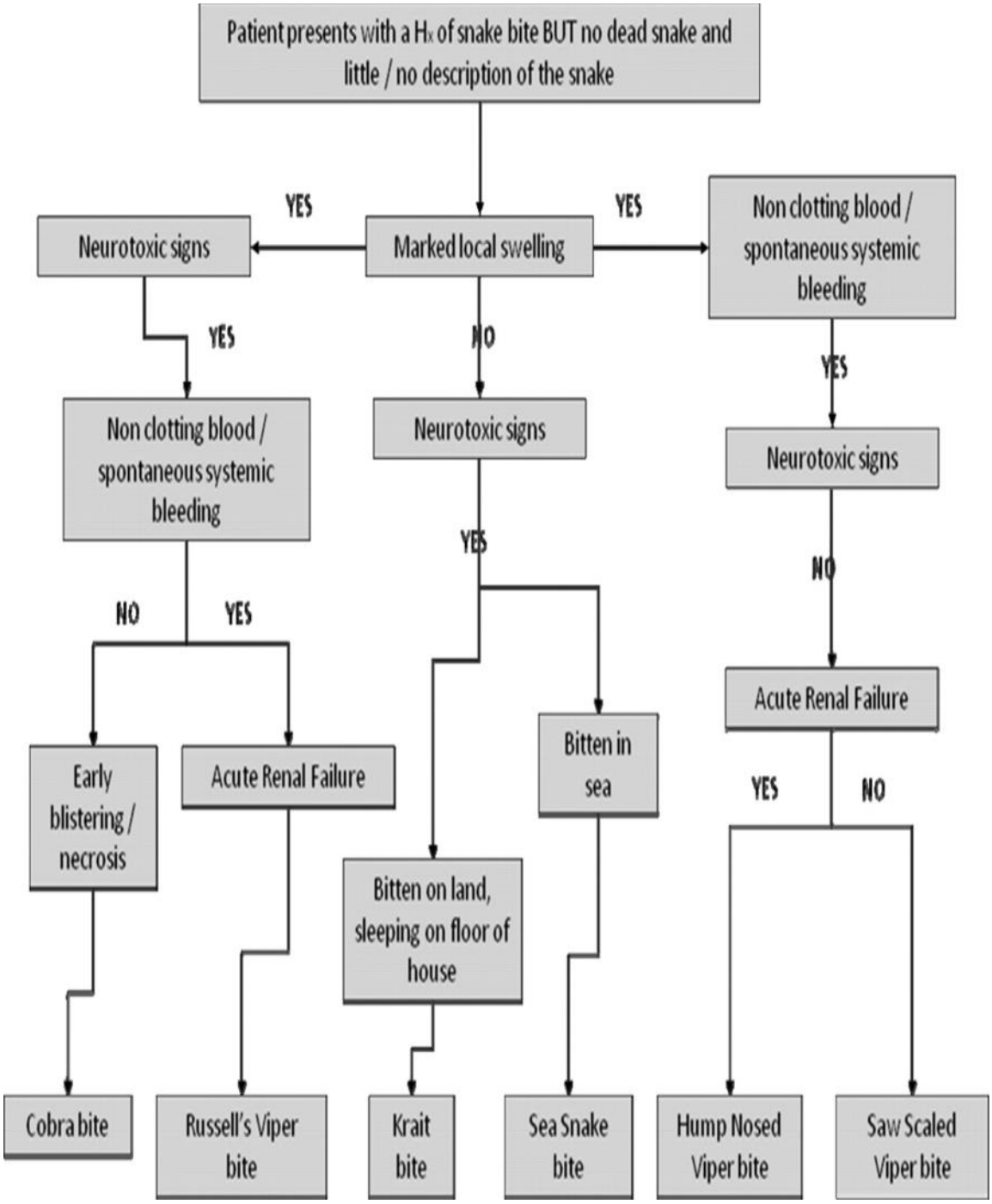
Haematological features:

Petechial and/or purpuric rash is seen most commonly with viperidae family[29]. The local area of bite may become devascularized with features of ischemia and necrosis predisposing to gangrene. Generally elapid bites result in early gangrene, usually wet type, whereas vipers cause dry

gangrene of slower onset (over weeks). It is caused mainly by direct cytotoxic venom effect. Massive haemorrhage which warrants transfusion are also common.Intra cerebral haemorrhage is a dreaded complication.

There are two case reports of Raynauds phenomenon and gangrene in a limb bitten by Russell's Viper[30], secondary infection including tetanus and gas gangrene may also result[31].

FIGURE 15: IDENTIFICATION OF BITE BASED ON SIGNS



Systemic Manifestations:

The most common and earliest symptom following snake bite is fright and anxiety, because of fear of dying .Many patients present symptoms which are direct result of fright irrespective of either poisonous or non poisonous bite. Patient may complain of sudden onset of weakness, defective vision ,difficulty in breathing ,dysphagia, syncopal attack, etc.

These symptoms have to be differentiated from that of neurotoxicity[32]. The time of onset of poisoning varies in different species, cobra produces symptoms as early as 6 to10 minutes, after bite, viper takes 7 to 15 minutes however symptoms may be delayed for several hours. Sea snake bites almost always produces myotoxic features within 3hours[33].

Elapid venoms produce paralytic effect but cobra bite commonly causes, severe local pain, swelling, blistering and tissue necrosis. Paralysis may be delayed for 7 – 12 hours after bite. These victims often presents with preparalytic symptoms like vomiting , blurred vision, drowsiness and tingling sensation around the mouth, paralysis first appears as bilateral ptosis and external ophthalmoplegia and then spreads to involve muscles of palate tongue, jaw, neck, larynx, and finally respiration. Finally

leading to generalized flaccid paralysis, consciousness is maintained through out, provided there is no cardiac or respiratory failure.

In general the viperidae venoms are best known for their severe local manifestations, haematotoxic effects and renal failure In none of the studies done in other countries was the presence of neurological manifestations recorded[36].

Some times Russells vipers systemic envenomation may develop in the absence of local reactions. . Russells viper venom activate factors V and X and cause fibrinolysis. Certain venoms cause defibrinogenation by activating endogenous fibrinolytic system[37,38.].Spontaneous systemic bleeding resulting from haemorrhagins, which damage vascular endothelium and some times the blood does not coagulate due to consumption coagulopathy.

All these effects result in bleeding from gum, epistaxis, haematuria, haematemesis and malena, kidney, from fang marks and even from vene puncture sites and some times as multiple petechiae and purpurae[39,40]. Intra cerebral haemorrhage (Subarcahnoid haemorrhages and cerebral haemorrhage) have also been reported[40].

Hypotension and shock mainly result from hypovolaemia due to massive haemorrhage or extravasation of fluid from the intra vascular compartment into the swollen limb. Vasodilation and a direct action on the myocardium is mainly seen in viper bite[41,42].

Almost every species of snakes can cause renal failure. It is fairly common following Russell's viper bite and is also a major cause of death[42]. Mechanism of renal damage include ischaemia (from hypotension, renal vasoconstriction or disseminated intravascular coagulation), haemorrhage, direct nephrotoxicity ,acute tubular necrosis , pigment nephropathy associated with massive intravascular haemolysis, generalized rhabdomyolysis and associated electrolyte disturbances[41].

Acute interstitial nephritis due to snake venom have also been observed[43], Cardiotoxic features, include tachycardia, hypotension or hypertension and ECG changes including sinus tachycardia, ischaemic non-specific ST-T changes, arrhythmias and artrio-ventricular blocks. This cardiotoxicity is seen in 25% of viperine bites[44].

Myalgic features are the most common presentation of bites by sea snakes. Muscle necrosis may also result in myoglobinuria. Rare systemic

manifestations including hypopituitarism[45], bilateral thalamic haematoma[46] have also been reported

Severity assessment.

Snakebite severity score (SSS) was given by Dart et al.,1996 and Nualnong et al., 2005[47,48]. The grading of severity was based on severity of symptoms or signs observed. The severity was graded from 0 to 4 ,ranging from no envenomation to severe life threatening symptoms and death taking into consideration clinical signs/symptoms and/or laboratory data.

The four levels represent as follows; grade 0 for no symptoms or signs, grade 1 for mild, transient and spontaneously resolving symptoms or signs, grade 2 for moderate, pronounced or prolonged symptoms or signs, grade 3 for severe or life threatening symptoms or signs, while grade 4 represents extremely severe envenomation leading to mortality. Occurrence of a particular symptom was checked against the chart and graded.

TREATMENT OF SNAKE BITE:

First Aid Management[49]

The majority of current first aid methods adopted by victims such as tourniquets, cutting ,biting and suction ,herbal remedies,application of heat are completely ineffective and dangerous. Current recommendation is to adopt what has been called the 'Do it R.I.G.H.T.' approach, stressing the need for Reassurance, Immobilisation as per a fractured limb, Getting to Hospital without delay and Telling the doctor of any symptoms that develop.

Support Posters and pamplets have been designed detailing this method and providing pictures of the major venomous species. It is recommended that these posters are given the widest possible distribution throughout the State in schools, community centres, hospitals and other public places.

1. **Reassurance**: The patient should be reassured by pointing out that all snakes are not poisonous, even poisonous snakes cannot always inject a lethal dose.
2. **Immobilize** the bitten limb with a splint. Avoid applying tourniquets or pressure bandages

3**. Getting** to Hospital without delay

4. **Telling** the doctor of any symptoms that develop.

Hospital Treatment:

Indication for Antivenom Treatment:

A) Systemic Envenoming:

1. Neurotoxicity (Ptosis, ophthalmoplegia ,diplopia etc.).
2. Incoagulable blood indicating consumption coagulopathy (DIC).
3. Spontaneous systemic bleeding like bleed from bite site ,gingival bleed ,epistaxis,hematuria,etc
4. Hypotension (shock).
5. Generalized rhabdomyolysis .
6. Impaired consciousness.

B) Severe, Local Envenoming:

1. Extensive local swelling (involving more than half of the bottom limb).
2. Rapidly evolving local swelling (extending beyond the bitten segment of limb, for example above the ankle in bite on the foot) within one hour of bite.
3. Bites on the digits (fingers and toes) where venom is known to cause local necrosis (e.g., viper and cobra).

Monospecific (monovalent) antivenom is ideal if the biting species is known, polyspecific (polyvalent) antivenom in India is effective against the four common varieties of snakes. It is prepared from the horse's serum hyper-immunized with the venom. It is seen that in large number of

envenomations the type of snake could not be identified. So in Indian situation, it is probably safer for the time being to use the polyvalent anti-snake venom (ASV).

The optimal therapy of poisonous snake bite remain controversial. The optimal dose of ASV, frequency of administration and duration of therapy remains unclear. There are no clearly defined end points for stopping ASV therapy in neurotoxic cases[50]. While treating viper bite cases one should not hesitate to use higher dose of ASV, if the coagulation parameter remains abnormal. Dose of ASV should be repeated (preferably every 6 hours) until coagulation time returns to normal.

Coagulation parameters should be repeated several times because value may be transiently normal and later on may become abnormal. This is probably due to continued release of venom from injection site and this warrants repeating ASV, as half life of ASV is 26 to 95 hours and initial lethal dose of ASV may not prevent late envenoming.

Swelling at the site of bite may be used as a possible indicator to give further dose of ASV, when there is a doubtful picture of coagulation abnormality[52,53]. Each one of polyvalent ASV neutralizes not less than the following quantities of standard venoms tested in mice by intravenous route.

- Cobra (Naja Naja) - 0.6 mg.
- Common krait (Bungarus Caerueus) - 0.45 mg

- Russells viper (Vipera Russelli) - 0.6 mg
- Saw scaled viper (Echis carinatus) - 0.45 mg

Dosage[53] :

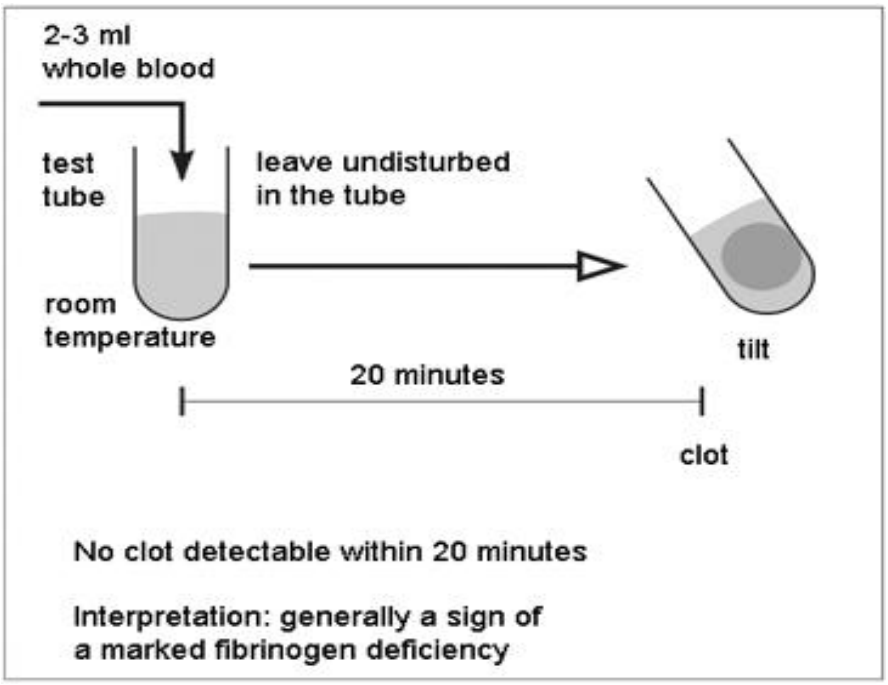
The current medical education in India is reliant on Western textbooks for snake bite management. This has led to inapplicable protocols being employed in particular the inappropriate administration of polyvalent anti snake venom (ASV) when it is not required and/or in doses well in excess of the required amount. The current protocol has been developed in the Indian context and with reference to Indian species.

Research has shown that PHCs do not treat snakebite mainly due to confidence issues and the ability of Primary Health Centres to deal with snakebite should be increased. The earlier an envenomed patient is treated with ASV, better the outcome.

ASV Administration Criteria[49]

The grounds for administering ASV have been rationalised to better suit the Indian context. It should be administered if there is significant envenomation i.e. incoagulable blood shown by the 20 WBCT or significant limb swelling for viperine bite, neurological signs for elapidae bite.

FIGURE 16 : 20 MINUTES WHOLE BLOOD CLOTTING TIME



ASV Dosage & Repeat Dosage

The recommended initial dose of ASV is 8-10 vials administered over 1 hour. Mode of administration is IV only

- Repeat doses for haemotoxic species is based on the 6 hour rule
- Repeat doses for neurotoxic is based on the 1-2 hour rule.
- The maximum recommended dose for haemotoxic bites in 30 vials of ASV
- The maximum recommended dose for neurotoxic bites is 20 vials of ASV

Neurotoxic Bite Support Measures

Despite the fact that the neostigmine test was actually an Indian discovery, it is still poorly used in India. The neostigmine test should be carried out in all neurotoxic bites But should be accompanied by objective measures of improvement. It is an all or nothing test. If there is no positive response to first dose, the neostigmine should be discontinued.

The Management of Snakebite in Primary and Community Care Hospitals

All PHC/CHCs are capable of managing the initial stages of snakebite if confident and equipped and with explicit referral criteria. Where the new protocol has been trialled this is becoming more evident.

A basic drug and equipment profile for the PHC/CHC has been proposed. This includes methods for calculating the ASV requirement. It should be noted that many States already have ASV in PHC/CHCs but doctor confidence is preventing usage.

Haemotoxic bites with correct signs of envenomation can be treated with 8-10 vials of ASV, if any ASV reaction occurs treat with adrenaline and then transferred to a higher centre with the ability to carry out the required blood tests to identify occult bleeding or renal impairment.

Neurotoxic bites with correct signs of envenomation can be treated with 8-10 vials of ASV, stabilised if any ASV reaction occurs with adrenaline and administered the neostigmine test. If there is no evidence of impending

respiratory failure, determined by patient ability to perform a neck lift the patient can be treated locally. If the patient is unable to perform a neck lift then they will be transferred to a higher centre with mechanical ventilatory capability. Transfer steps including airway support are specified.Support Posters have been prepared in support of the Protocol for use at the bedside in PHC/.CHCs, District Hospitals and Tertiary centres

It is not advisable to inject venom antiserum at the local site of skin bite.

ASV REACTIONS:

Antivenom can cause two types of reaction, an early (anaphylactoid reaction) and a late (serum sickness type) reaction. The early (anaphylactoid) reaction is characterized by itching, urticaria, cough, nausea, vomiting, fever and tachycardia. Some may develop systemic anaphylaxis with hypotension, bronchospasm and angioneurotic oedema. This is treated with adrenaline 0.5 to 1.0 ml subcutaneously (may be repeated), chlorpheniramine maleate 10 mg IV and hydrocortisone 100 mg IV[54,55]

MANAGEMENT OF SHOCK:

If the patient is hypovolaemic, indicated by supine or postural hypotension, empty neck veins, sunken eyeballs, loss of skin turgor and dryness of mucosae, proceed as follows:

1) Establish intravenous access

2) Bladder catheterisation

3) Central venous pressure monitoring :

This can be achieved **either** by observing the vertical height of the jugular venous pulsation above the sternal angle with the patient propped up on pillows at 45^{o}; **or** by direct measurement of central venous(superior vena caval) pressure through a long catheter preferably inserted at the antecubital fossa.

The catheter is connected to a saline manometer, the 0 point of which must be placed at the same level as the right atrium (that is, at the sternal angle when the patient is propped up at 45^{o}). In someone who is obviously volume-depleted, resuscitation should start immediately, and not be delayed until a central venous line has been inserted.

4) Fluid challenge:

Depending on the initial state of hydration/dehydration, an adult patient can be given two litres of isotonic saline over one hour or, until the jugular venous pressure/central venous pressure has risen to 8-10 cm above the sternal angle (with the patient propped up at 45^{o}). The patient must be closely observed while this is being done. The fluid

challenge must be stopped immediately if pulmonary oedema develops. If the urine output does not improve, try furosamide challenge.

5) Furosamide challenge:

60-100 mg of furosamide is injected slowly (4-5 mg/minute). If this does not induce a urine output of 40 ml/hour, give a second dose of furosamide, 200 mg. If urine output does not improve, try mannitol challenge.

6) Mannitol challenge:

175 ml of 20% mannitol may be infused intravenously over 15 minutes but this must not be repeated ,as there is a danger of inducing dangerous fluid and electrolyte imbalance. An improvement in urine output to more than 40 ml/hr or more than 1 litre/day is considered satisfactory.

7) Conservative management:

If the patient is still oliguric, despite these challenges, diuretics should be stopped and fluid intake should be restricted to a total of the urine output plus "insensible losses" (500-800 ml/day). If possible, the patient should be referred to a nephrology unit. The diet should be bland, high in calories (1800/day), low in protein (less than 40g/day), low in potassium (avoid citrus fruit, fruit juices and potassium-containing drugs) and low in salt. Infections will lead to tissue breakdown and increase urea levels. So infections should be

prevented or treated promptly with non-nephrotoxic antibiotics (ie avoid aminoglycosides such as gentamicin).

8) Biochemical monitoring:

Serum potassium, urea, creatinine and, if possible, pH, bicarbonate, calcium and phosphate should be monitored frequently. If this is not possible the electrocardiogram (ECG) should be examined for evidence of hyperkalaemia, especially following bites by sea snakes, or Sri Lankan or South Indian Russell's vipers or if the patient is passing dark brown urine, indicating rhabdomyolysis or intravascular haemolysis

Compartment syndrome and fasciotomy

The presence of an immobile, swollen,shiny, cold and apparently pulseless snake-bitten limb may suggest the possibility of increased intracompartmental pressure, especially if the digital pulp spaces or the anterior tibial compartment are involved. Swelling of affected muscle within tight fascial compartments will result in an increase in tissue pressure above the venous pressure, resulting in ischaemia. However, the classical signs of an intra compartmental pressure syndrome may be difficult to assess in snakebite victims.

Clinical features of compartment syndrome

1) Disproportionately severe pain

2) Weakness of intracompartmental muscles

3) Pain on passive stretching of intracompartmental muscles

4) Hypoaesthesia of areas of skin supplied by nerves running through the compartment

5) Obvious tenseness of the compartment on palpation

Criteria for fasciotomy in snake-bitten limbs

1) Haemostatic abnormalities have been corrected (antivenom with or without clotting factors)

2) Clinical evidence of an intracompartmental syndrome

3) Intracompartmental pressure >40 mmHg (in adults)

Rehabilitation :

Restoration of normal function in the affected part after the patient has been discharged from hospital is not usually supervised. Routine physiotherapy may help to accelerate this process. In patients with severe local envenomation , the limb should be maintained in a elevated position. In order to prevent the deformity leg should be kept

in functional position .For example, in the leg, deformity of the ankle can be prevented by application of a back slab.

FIGURE 17:FASCIOTOMY DONE IN A PATIENT WITH COMPARTMENTAL SYNDROME

Other Measures:

1. Respiratory paralysis should be treated by artificial ventilation.
2. Hypotension and shock should be treated with fresh whole blood or fresh frozen plasma, dopamine and hydrocortisone.
3. Oliguria and renal failure should be treated conservatively failing which dialysis should be done.
4. Local infection should be prevented by antibiotics covering gram negative organisms and anaerobes. All patients should receive antitetanus prophylaxis.

5. At a later stage some patients may require surgical debridement, skin grafting or fasciotomy.
6. Haemostatic disturbance should be treated with fresh whole blood or fresh frozen plasma or platelet concentrates.

Do Not's in Snake Bite:

- Local incision and suction of wound.
- Application of ice packs and potassium permanganate.
- Application of tight constriction bands impeding arterial flow.
- Infiltration of antivenom locally.

COMPLICATIONS OF SNAKE BITE:

1. **Respiratory Failure**: In cases of severe poisoning by Elapid snakes there can be paralysis of the intercostal muscles and diaphragm leading to respiratory failure. Patients may have confused, drowsy, stuporous or even comatose. The respiratory failure is of type-II i.e., there is both arterial hypoxia as well as hypercapnia.
2. **Shock**: One of the most important complications of envenomation is shock due to large amount of peptide and protein content of the venom. Patients may show signs of peripheral circulatory failure with or without signs of impending coma, hypotension, renal failure, dehydration and electrolyte disturbances

3. Renal Failure: This is particularly seen in case of severe envenomation following bites by vipera Russelli. The cause of renal failure is due to ischaemia ,haemorrhage into the kidney, resulting from hypotension, disseminated intravascular coagulation and renal vasoconstriction. The renal cortical necrosis is due to the schwartzman like phenomenon[56], patients may have a decreased urine output of less than 400 ml/ 24 hours associated with fluid and electrolyte imbalance.

4. Gangrene:

Neglected local necrosis can result in gangrene of the limb. So the wound should be examined frequently for evidence of necrosis. Early signs of necrosis include blistering, blackening of the skin, loss of sensation and a characteristic smell of putrefaction. There is high risk of secondary infection and so the necrotic tissue should be debrided under local or general anesthesia as soon as possible.

Haematological alterations following viperidae snake venom poisoning:

- **Haemoglobin**: In systemic viper poisoning, anaemia caused by loss of red cells into the bitten limb and haemorrhage may result in a fall in haemoglobin of over 8 gm/ 100 ml[57].

- **Bleeding and clotting time**: In most cases of envenomation, bleeding and clotting times will be prolonged. A 20 minutes whole blood clotting test is a simple and extremely sensitive bedside test of systemic envenoming. With venom producing non-clotting blood , this always precedes abnormal bleeding, thus there can be non-clotting blood without abnormal bleeding but the reverse, abnormal bleeding with normal clotting is not observed.

- **Platelet count**: Platelet count may fall during the first few days after the bite, later it will return to normal level. Hess's test is usually positive.

- **Plasma fibrinogen**: Plasma fibrinogen is reduced in amount. There is continuous microcoagulation of fibrinogen with virtually simultaneous disposal of resulting fibrin.

- **Fibrin degradation product**: It's concentration is increased in blood and urine

- **Factors V and VIII**:

  The levels are reduced.

- **Coagulation profile**: prothrombin time and activated partial thromboplastin time are usually prolonged.

- **Prothrombin time**: It is usually prolonged.

PREVENTION:

Following tips will help one to escape snake bite[58]:

1. Avoid putting your limbs in places where your vision can't reach.

2. Do not handle snakes which are just killed and dying.

3. Avoid work at night in an endemic area without footwear. Farmers and agriculture based workers are motivated to use low cost foot wears.

4. The area and the surroundings of any human habitation or working fields should be kept clear of all ,bushes, herbs, garbage and rubbish to prevent sheltering of snakes.

5. Food should be kept away from sleeping place. It attracts rodents which in turn attract snakes.

6. Clean the floor under good light source before going to bed , especially in homes with mud floors

7. If a snake is encountered, it is safer to keep perfectly still. The snakes movement then could be judged and it can be distracted by throwing down some object.

Other measures to reduce incidence of mortality and morbidity:

1. Always take the patient immediately to the doctor rather than restoring to mantras or any other traditional treatment by non-medical persons. Public health education is the most important to emphasize about this.
2. All primary health centres should be fully equipped with facilities to give first aid measures and administer specific antisnake venom if required.
3. As snake bite is primarily a rural occupational hazards, educating the people regarding readily available effective treatment may drastically bring down the morbidity and mortality associated with treatment by quacks.

If the precautions mentioned above are observed then the incidence of snakebite and its morbidity and mortality can be reduced considerably[59]

METHODOLOGY

This study was carried out from June 2013 to May 2014(one Year Study), the study was conducted prospectively . The study comprised of 178 cases of snakebite poisoning admitted to the Department of General medicine , Chengalpattu Government Hospital.

Objectives of the Study:

1. To know the incidence of snake bite poisoning, time and site of bite and incidence among various sex and age groups and seasonal variation among patients admitted with snake bite poisoning in department of General Medicine.
2. To study the clinical presentation, complications, treatment and outcome of snake bite envenomation in adults.
3. To study the risk factors associated with complications.
4. To suggest measures to prevent deaths from snakebite poisoning.

The patients were studied, from the time of admission and followed up till discharge from the hospital or till death.

The data was collected from:

1. Preformed systematic questionnaire
2. Information gathered from treating Doctors in respective Medical units

3. Information from the victims or the relatives present regarding socioeconomic status, family history, time of bite, type of snake, and first aid measures taken.

The statistical analysis of the data collection was done and presented as results and observations in tabular form and graphs and charts.

Inclusion criteria:

1. All patients with signs and symptoms of snake bite envenomation of either sex of age >13 years admitted to Medicine Department .

At the time of admission enquiry is made about the type of snake, time of snakebite, site of snakebite, the signs and symptoms, and any first aid measures taken. Inquiry was also made about the preliminary data of the victim, such as age and occupation. In most of the cases, the history is elicited by the patient himself but in certain cases by the close relatives or by the accompanying people.

General Physical Examination:

The vital signs like pulse rate, blood pressure, respiratory rate and level of consciousness were noted. The site of the snakebite was examined for any local tissue reaction, such as swelling, erytherma, necrosis.

An attempt was made to correlate the pattern of fang marks with the type of alleged snake

Systemic Examinations:

A detailed examination of central nervous system, cardiovascular system, respiratory rate and per abdomen was carried out in all the cases.

Laboratory Investigations:

The blood was sent for the routine haematological investigations with a detail study of coagulation profile.

The treatment was instituted as early as possible:

1. Immediate resuscitative measures
2. Administration of anti-snake venom.
3. Symptomatic treatment

Exclusion Criteria:

1. Patients admitted for snake bite but without signs of envenomation .
2. Patients admitted with history of unknown bites or other bites without signs of snake bite envenomation

RESULTS

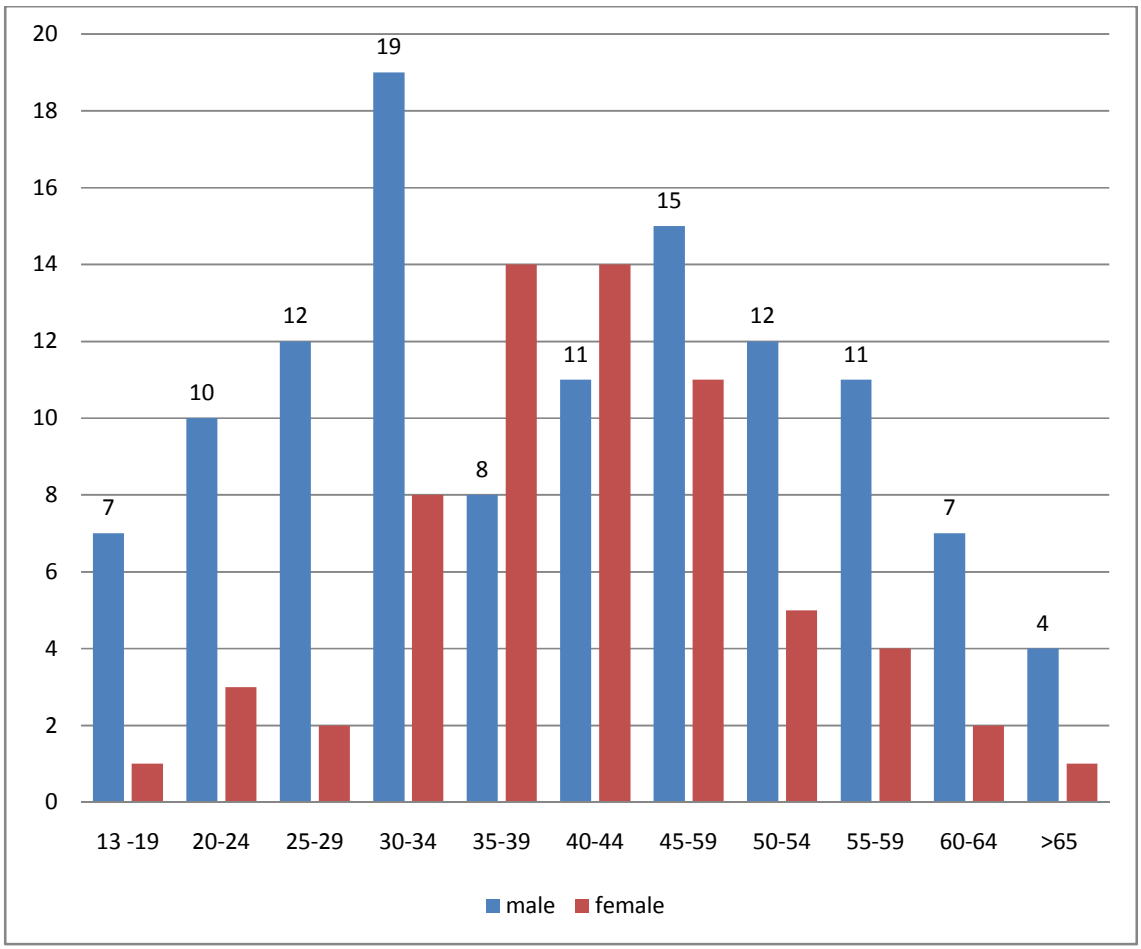
Table No. 1:- AGE Vs SEX WISE DISTRIBUTION OF STUDY POPULATION

AGE (years)	MALE		FEMALE		TOTAL	
	Number	Percent	Number	Percent	Number	Percent
13 -19	7	6.03	1	1.54	8	4.42
20-24	10	8.62	3	4.62	13	7.18
25-29	12	10.34	2	3.08	14	7.73
30-34	19	16.38	8	12.31	27	14.92
35-39	8	6.90	14	21.54	22	12.15
40-44	11	9.48	14	21.54	25	13.81
45-59	15	12.93	11	16.92	26	14.36
50-54	12	10.34	5	7.69	17	9.39
55-59	11	9.48	4	6.15	15	8.29
60-64	7	6.03	2	3.08	9	4.97
>65	4	3.45	1	1.54	5	2.76
TOTAL	116	100	65	100	181	100

χ^2	0.000101
As per table	18.307
Degree of freedom	10

$P<0.05$

Figure No.18:- AGE Vs SEX WISE DISTRIBUTION OF STUDY POPULATION



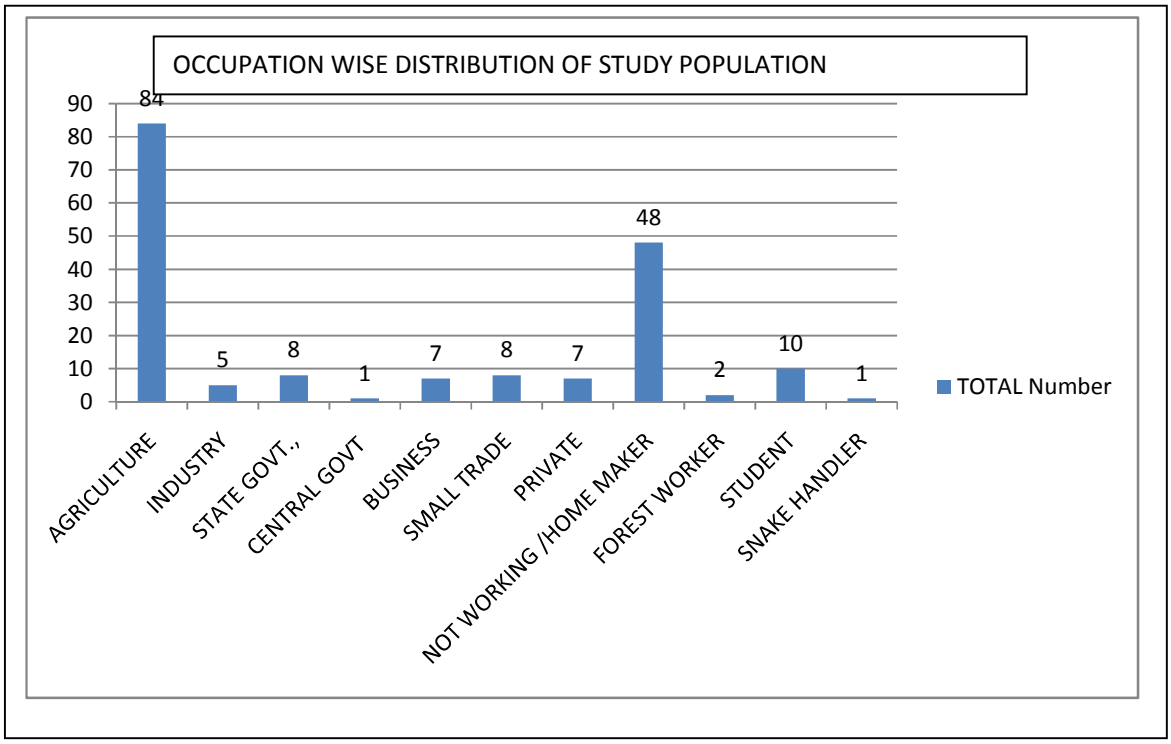
Among 181 cases, maximum number is 27 (14.92%) belong to 30-34 years of age Around 26.8%of cases belongs to fourth decade. Least number of cases 5(2.76%) belongs to >65 years of age. In the study of population males were 64.1% and females were 35.91% An significant association was found between age and sex study of population.

TABLE 2:- OCCUPATION WISE DISTRIBUTION OF STUDY POPULATION

OCCUPATION	MALE		FEMALE		TOTAL	
	Number	Percent	Number	Percent	Number	Percent
AGRICULTURE	72	62.07	12	18.46	84	46.41
INDUSTRY	1	0.86	4	6.15	5	2.76
STATE GOVT.,	7	6.03	1	1.54	8	4.42
CENTRAL GOVT	0	0.00	1	1.54	1	0.55
BUSINESS	3	2.59	4	6.15	7	3.87
SMALL TRADE	5	4.31	3	4.62	8	4.42
PRIVATE	3	2.59	4	6.15	7	3.87
NOT WORKING /HOME MAKE	13	11.21	35	53.85	48	26.52
FOREST WORKER	2	1.72	0	0.00	2	1.10
STUDENT	9	7.76	1	1.54	10	5.52
SNAKE HANDLER	1	0.86	0	0.00	1	0.55
TOTAL	116	100	65	100	181	100

$P<0.05$
So, the hypothesis is accepted

FIGURE 19 : OCCUPATION WISE DISTRIBUTION OF STUDY POPULATION



Out of 181 cases of snake bite , majority of cases i,e 46.41% are agriculture labourers .One case is a snake handler . Around 48 cases (26.52%) are not working or home make.

TABLE NO. 3:- IDENTIFIED SNAKE Vs UNIDENTIFIED SNAKE WISE DISTRIBUTION OF STUDY POPULATION

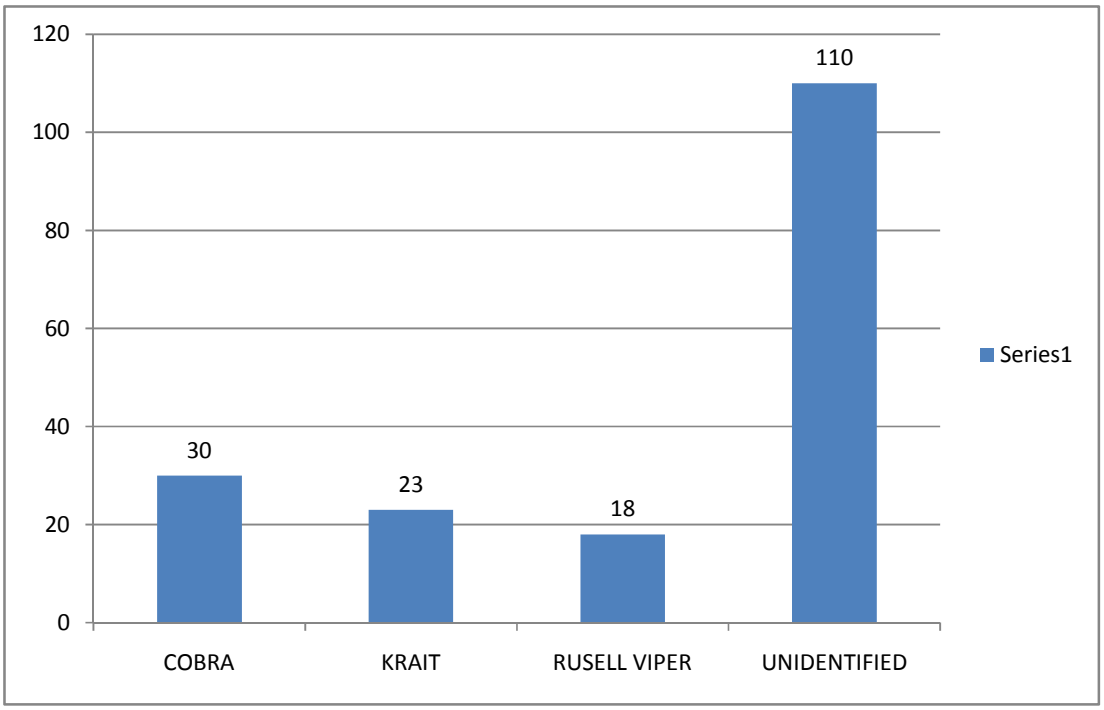
TYPE OF SNAKE	FEMALE		MALE		TOTAL	
IDENTIFIED SNAKE NAME	NO.	PERCENTAGE	NO.	PERCENTAGE	NO.	PERCENTAGE
COBRA	10	15.38	20	17.24	30	16.57
KRAIT	11	16.92	12	10.34	23	12.71
RUSELL VIPER	3	4.62	15	12.93	18	9.94
UNIDENTIFIED	41	63.08	69	59.48	110	60.77
TOTAL	65	100.00	116	100	181	100.00

χ^2	0.224259182301467
As per table	7.815
Degree of freedom	3

$P<0.05$

So, the hypothesis is accepted

FIGURE 20 :- IDENTIFIED SNAKE vs UNIDENTIFIED SNAKE WISE DISTRIBUTION OF STUDY POPULATION



Out of 181 cases 110 cases did not identify the snake . Of remaining 71 cases 16.57 percent of identified snake was cobra , followed by krait (12.71%) and rusell viper (9.94%)

TABLE NO. 4 :- COMORBIDS DISTRIBUTION OF STUDY POPULATION

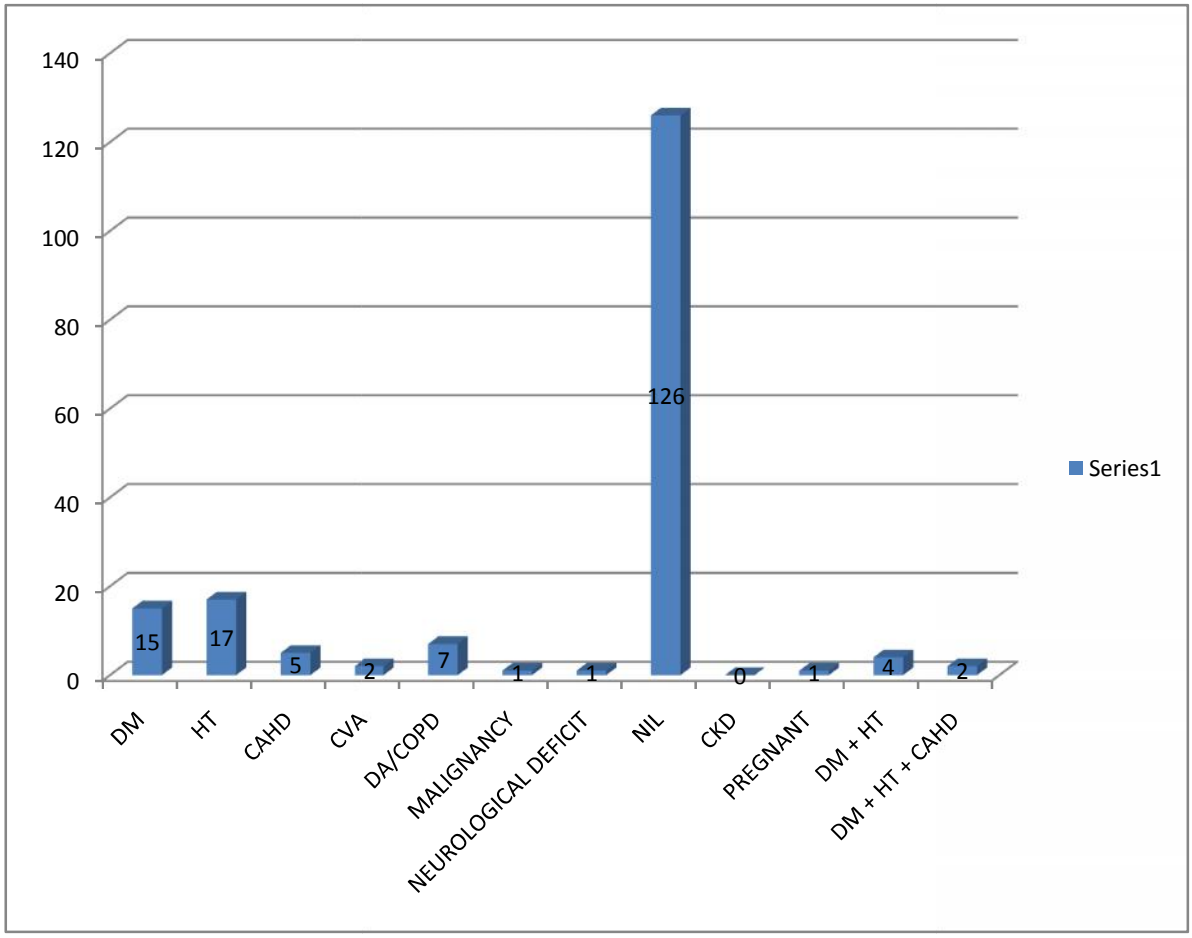
COMORBIDS	MALE		FEMALE		TOTAL	
	No.	PERCENTAGE	No.,	PERCENTAGE	No.,	PERCENTAGE
DM	8	6.90	7	10.77	15	8.29
HT	11	9.48	6	9.23	17	9.39
CAHD	2	1.72	3	4.62	5	2.76
CVA	2	1.72	0	0.00	2	1.10
BA/COPD	6	5.17	1	1.54	7	3.87
MALIGNANCY	0	0.00	1	1.54	1	0.55
Residual polio	1	0.86	0	0.00	1	0.55
NIL	80	68.97	46	70.77	126	69.61
CKD	0	0.00	0	0.00	0	0.00
PREGNANT	0	0.00	1	1.54	1	0.55
DM + HT	4	3.45	0	0.00	4	2.21
DM + HT + CAHD	2	1.72	0	0.00	2	1.10
TOTAL	116	94.82758621	65	98.46153846	181	96.13259669

χ^2	1.2562E-188
As per table	19.765
Degree of freedom	11

$P<0.05$

So, the hypothesis is accepted

FIGURE 21 :- COMORBIDS DISTRIBUTION OF STUDY POPULATION

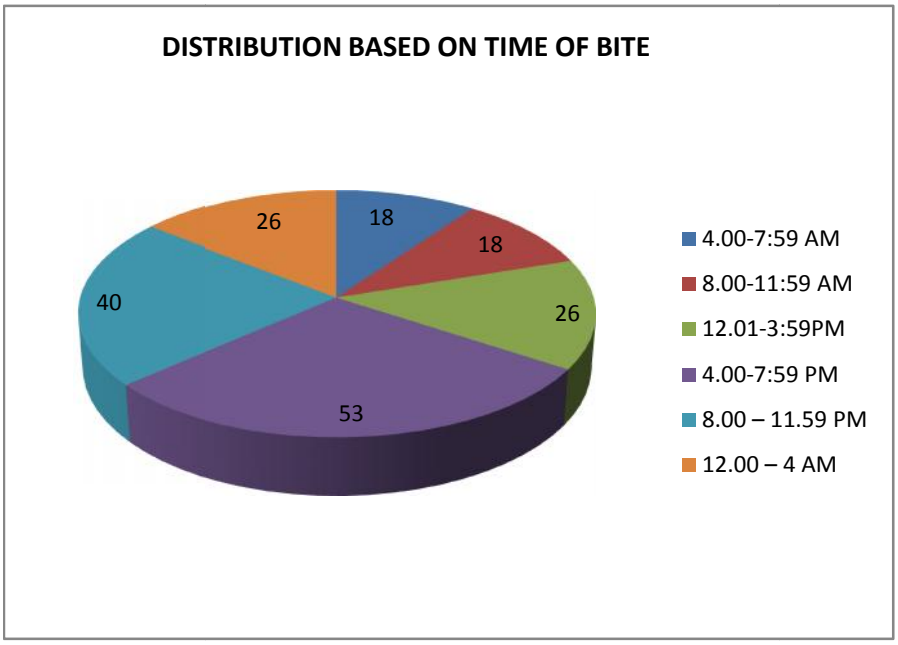


Among study population 55 patients have associated co-morbid illness with around 9.39 percent of patients are hypertensive , 8.29 percent are diabetic and one patient had old history of Breast carcinoma , 1 patient was an antenatal mother . 2 patients on treatment for DM,SHT and CAHD .

TABLE 5 : DISTRIBUTION BASED ON TIME OF BITE

TIME OF BITE	NUMBER	PERCENT
4.00-7:59 AM	18	9.94
8.00-11:59 AM	18	9.94
12.01-3:59PM	26	14.36
4.00-7:59 PM	53	29.28
8.00 – 11.59 PM	40	22.10
12.00 – 4 AM	26	14.36
TOTAL	181	100

FIGURE 22 : DISTRIBUTION BASED ON TIME OF BITE



Out of 181 , 53 (29.28%) cases ,the time of bite was evening time between 4.00 pm to 7.59 pm . Least number of bites(9.94%) are distributed in morning hours i,e 4 to 7.59 am and 8 to 11.59 am . A significant association was found between time of bite and study population

TABLE 6 :SITE OF BITE DISTRIBUTION

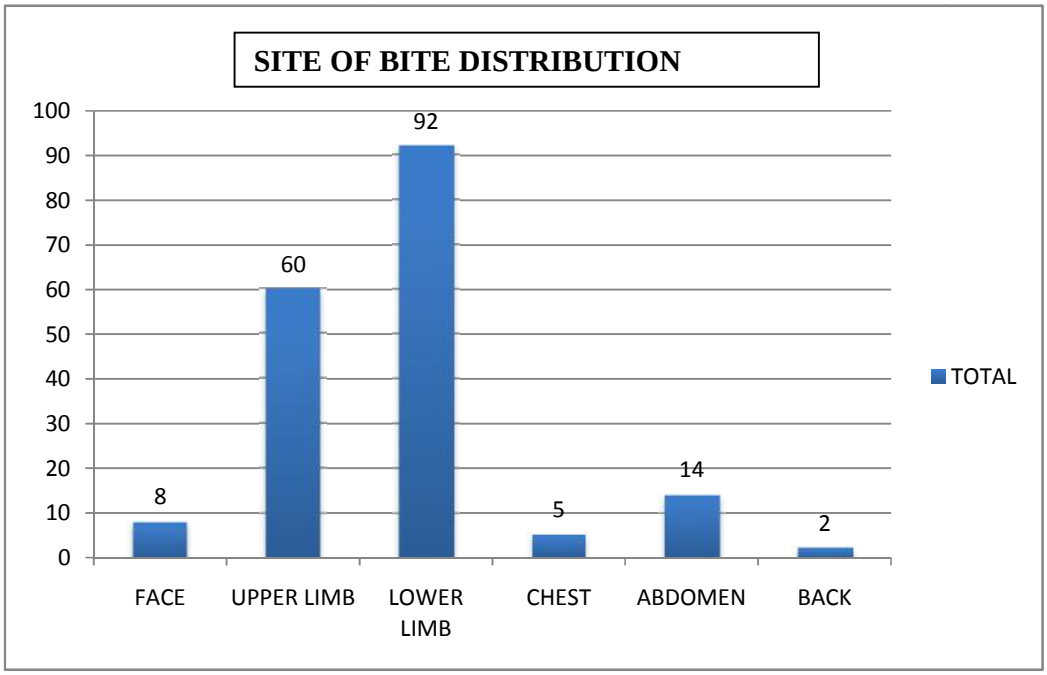
SITE OF BITE	MALE	%	FEMALE	%	TOTAL	%
FACE	7	6.03	1	1.54	8	4.42
UPPER LIMB	40	34.48	20	30.77	60	33.15
LOWER LIMB	59	50.86	33	50.77	92	50.83
CHEST	5	4.31	0	0.00	5	2.76
ABDOMEN	4	3.45	10	15.38	14	7.73
BACK	1	0.86	1	1.54	2	1.10
TOTAL	116	100	65	100	181	100

χ^2	2.19605E-47
As per table	11.07
Degree of freedom	5

$P<0.05$

So, the hypothesis is accepted

FIGURE 23 :SITE OF BITE DISTRIBUTION



Among 181 cases , 92 cases (50.83 %) were bitten in lower limb followed by 60(33.15%) of cases were bitten in upper limb . A significant association was found between site of bite and study population.

TABLE 7:PLACE OF BITE DISTRIBUTION

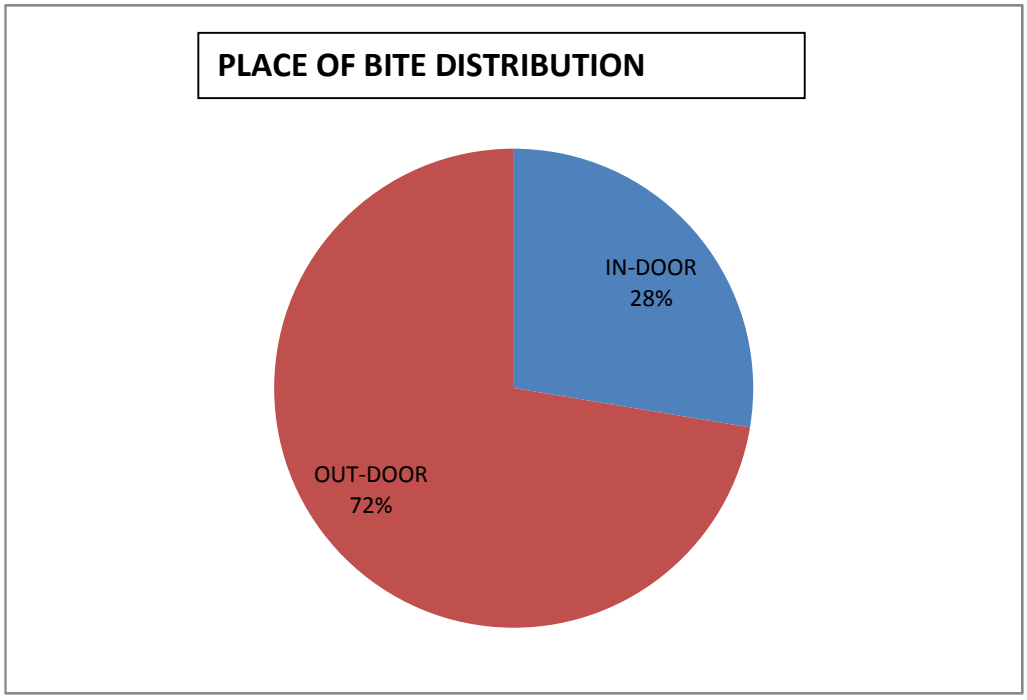
SITE OF BITE	MALE	PERCENT	FEMALE	PERCENT	TOTAL	PERCENT
IN-DOOR	26	22.41	24	36.92308	50	27.62
OUT-DOOR	90	77.59	41	63.07692	131	72.38
TOTAL	116	100	65	100	181	100

χ^2	1.73683E-09
As per table	3.841
Degree of freedom	1

$P<0.05$

So, the hypothesis is accepted

FIGURE 24 :PLACE OF BITE DISTRIBUTION



About 131(72 %)cases are outdoor (farm,work place, school ground,forest) and 50(28%) are indoor bite (home, other buildings). A significant association was found between place of bite and study population.

TABLE 8:DISTRIBUTION OF POPULATION BASED ON TOXICITY

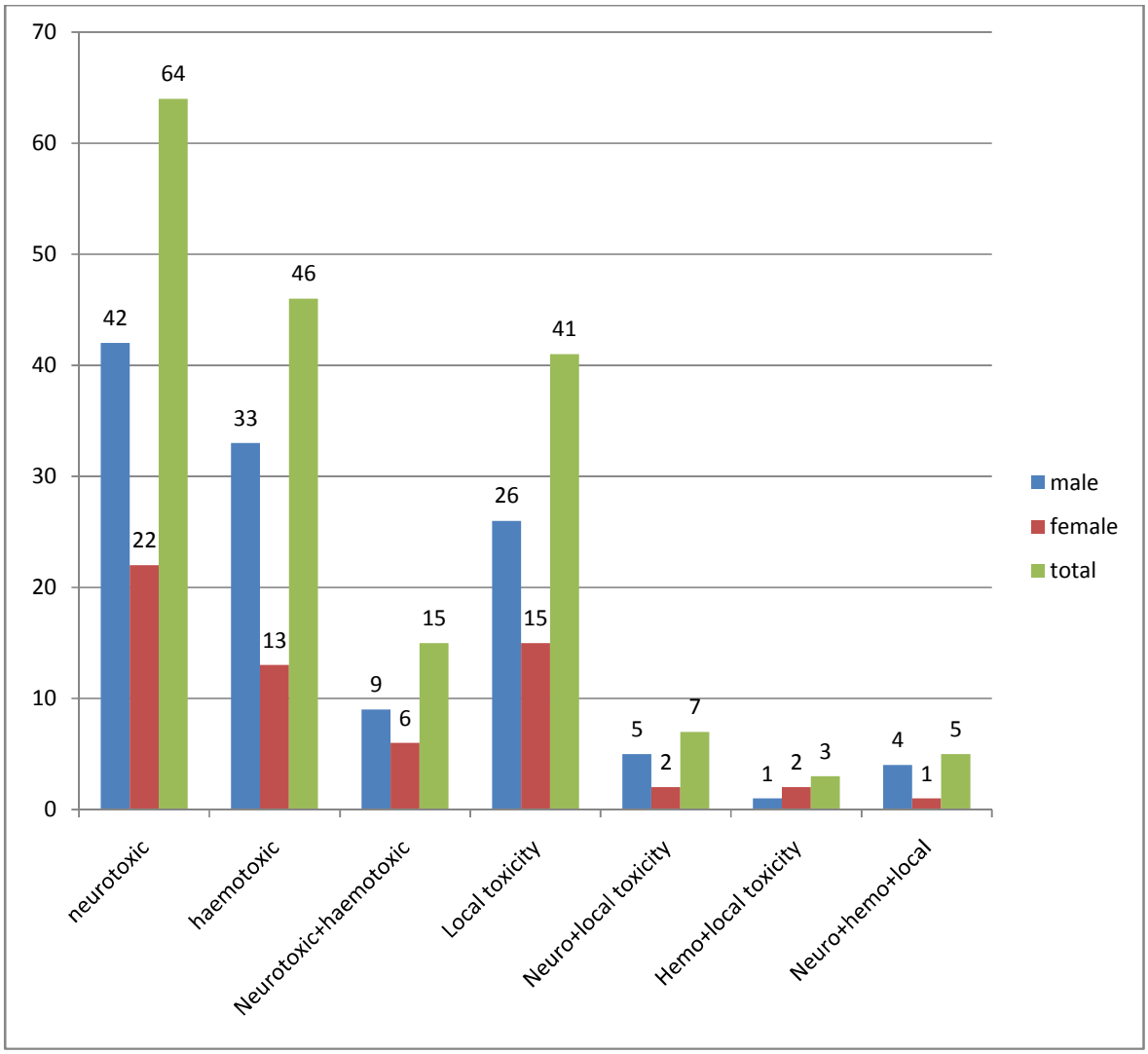
Toxicity	Male		Female		Total	
	Number	%	Number	%	Number	Percent
Neurotoxic	42	65.62	22	34.38	64	35.36
Haemotoxic	33	71.74	13	28.26	46	25.42
Neurotoxic+haemotoxic	9	60	6	40	15	8.28
Local toxicity	26	63.41	15	36.59	41	22.65
Neuro+local toxicity	5	71.43	2	28.57	7	3.87
Hemo+local toxicity	1	33.33	2	66.67	3	1.66
Neuro+hemo+local	4	80	1	20	5	2.76

χ^2	1.73683E-09
As per table	5.6821
Degree of freedom	6

$P<0.05$

So, the hypothesis is accepted

FIGURE 25 :DISTRIBUTION OF POPULATION BASED ON TOXICITY



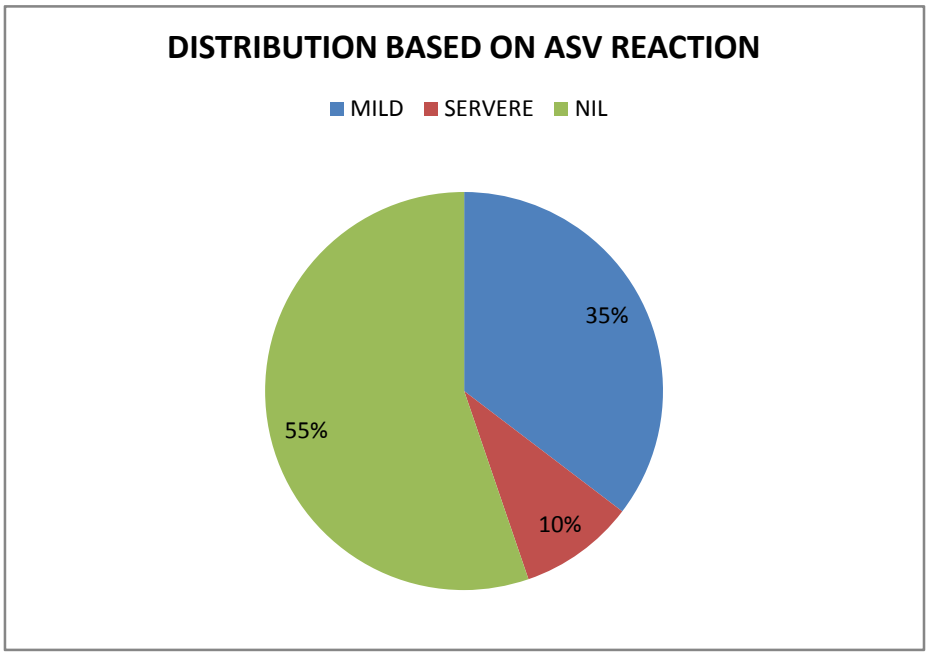
Neurotoxicity(35.38) is high among the toxicities observed . Next comes the pure haemo toxicity (25.42%) . Local reactions like cellulitis ,edema comprises 22.65 % .

TABLE 9: DISTRIBUTION BASED ON ASV REACTION

ASV REACTION	MALE	PERCENT	FEMALE	PERCENT	TOTAL	PERCENT
MILD	40	34.48	24	36.92	64	35.36
SERVERE	14	12.07	3	4.62	17	9.39
NIL	62	53.45	38	58.46	100	55.25
TOTAL	116	100	65	100	181	100

χ^2 - 3.385E-13 $P<0.05$

FIGURE 26 : DISTRIBUTION BASED ON ASV REACTION



Out of 181 cases 100 patients(55%) showed no reaction to ASV while in remaining 81 cases , 64 (35%) developed minor reactions in the form of fever , utricarial rashes and rigors . 17 cases (10%) developed severe anaphylactic reaction

TABLE 10 :DURATION OF STAY WISE DISTRIBUIION

DURATION OF STAY	NUMBER	PERCENT
<1 day	16	8.84
1-5days	108	59.67
5-10days	41	22.65
10-15days	14	7.73
>15 days	2	1.11

Of 181 cases maximum number of cases (59.67%) , number of hospital stay is less than 5 days . Only 16 cases are discharged with in 1 day after treatment .

FIGURE 27 : DURATION OF STAY WISE DISTRIBUTION

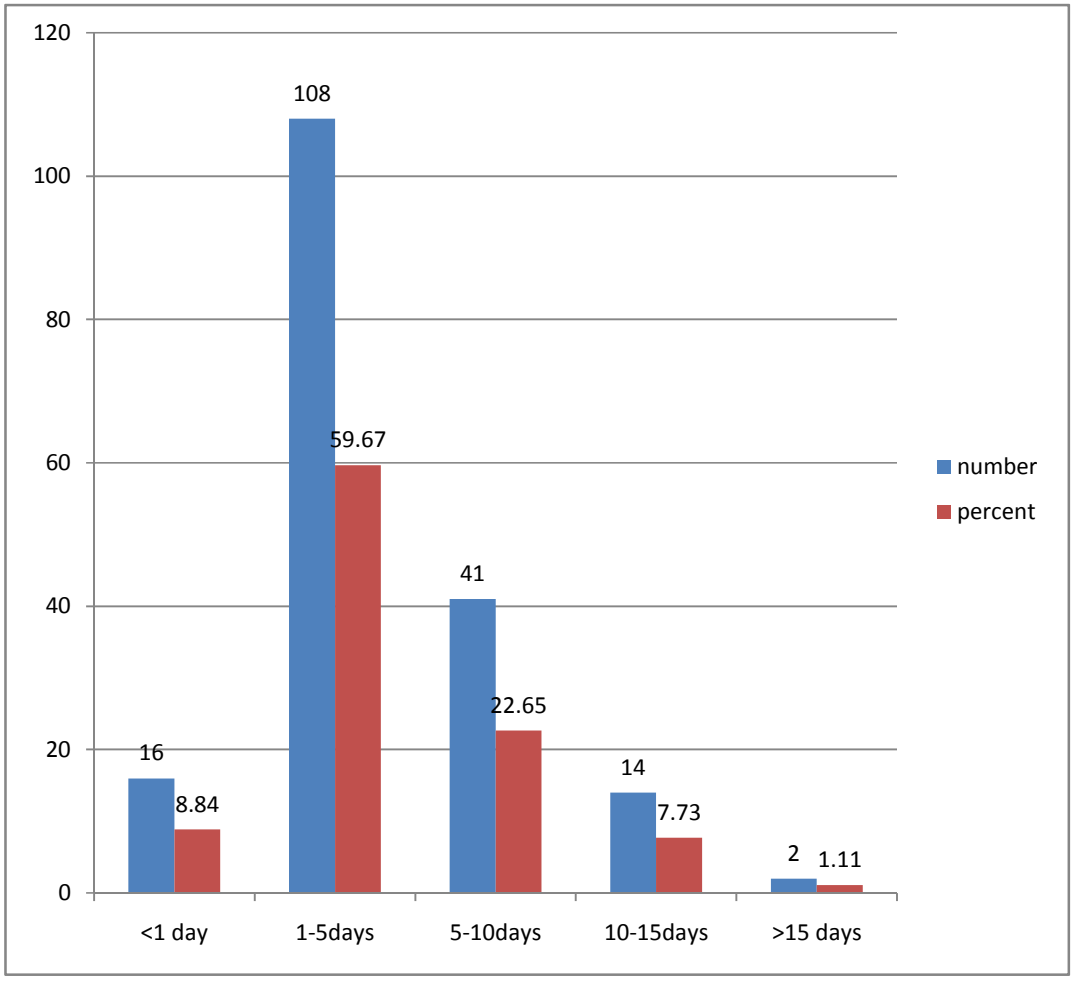
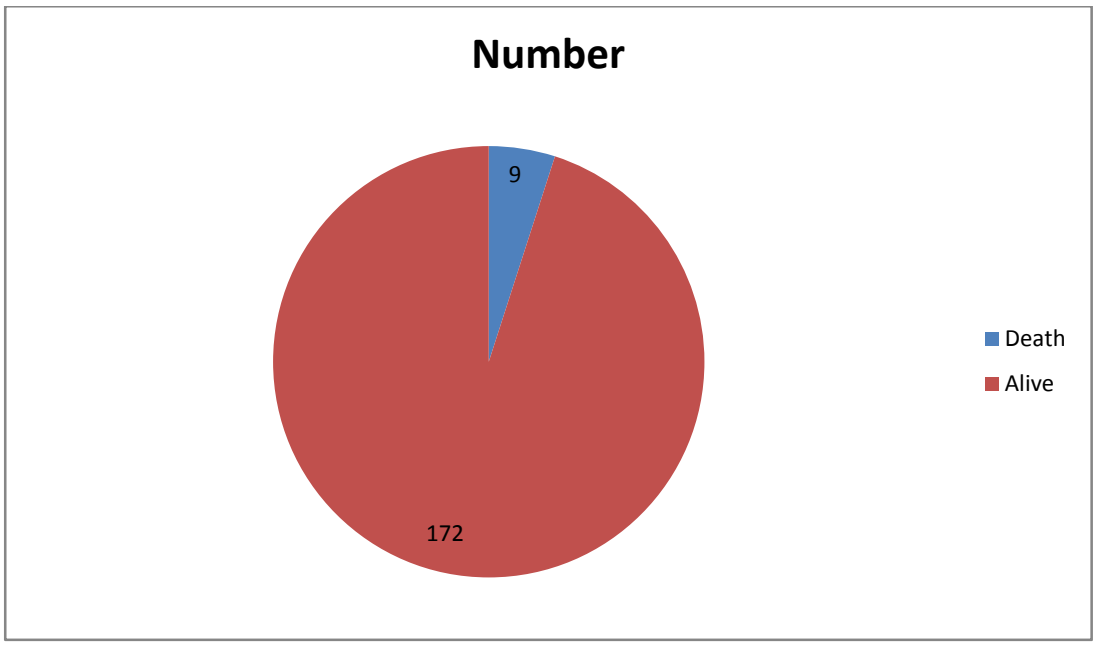


TABLE 11 DEATH Vs ALIVE

Outcome	Number	Percent
Death	9	4.97
Alive	172	95.03
Total	181	100

9 out of 181 are dead , 172 patients were alive .

FIGURE 28 :DEATH VS ALIVE



Out of 181 patients 9 patients (4.97)were dead .172 patients (95.03) were alive .

TABLE 12 :DISTRIBUTION BASED ON NATIVE TREATMENT VS ALIVE

NATIVE TREATMENT	FEMALE		MALE		TOTAL	
	NO	%	NO	%	NO	%
TOURNIQUETT	20	32.26	28.00	25.45	48	27.91
NIL NATIVE TREATMENT	41	66.13	76.00	69.09	117	68.02
OTHER	1.00	1.61	6.00	5.45	7.00	4.07
TOTAL	62.00	100.00	110.00	100.00	172.00	100.00

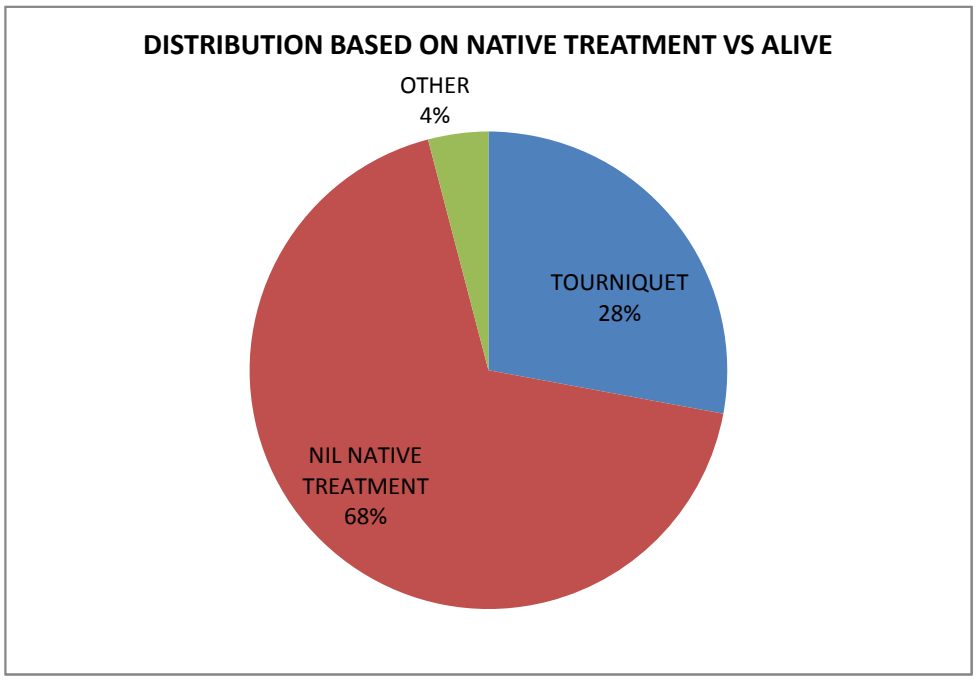
χ^2	3.66762E-33
As per table	7.815
Degree of freedom	3

$P<0.05$

So, the hypothesis is

accepted

FIGURE 29 :DISTRIBUTION BASED ON NATIVE TREATMENT VS ALIVE



In patients those who were alive ,history of tourniquet application was present in 48 (27.91%) cases, 7 cases (4.07%) had history of other methods of native treatment like cutting ,suction,etc. About 117 cases didnt recieve any native treatments .

A significant association was found between tourniquet application ,other native treatments and study population

TABLE 13: NATIVE TREATMENT vs MORTALITY WISE DISTRIBUTION OF STUDY POPULATION

NATIVE TREATMENT	FEMALE		MALE		TOTAL	
	NO	%	NO	%	NO	%
TOURNIQUET	3.00	100.00	6.00	100.00	9.00	100.00
NIL NATIVE TREATMENT	0.00	0.00	0.00	0.00	0.00	0.00
OTHER	0.00	0.00	0.00	0.00	0.00	0.00
TOTAL	3.00	100.00	6.00	100.00	9.00	100.00

Out of 9 patients who were dead ,history of native treatment in the form tourniquet application , wound cutting, suction etc were present in all cases

TABLE NO. 14:- BITE TO NEEDLE TIME VS MORTALITY WISE DISTRIBUTION OF STUDY POPULATION

BITE TO NEEDLE TIME	MALE		FEMALE		TOTAL	
	NO. OF CASES	%	NO. OF CASES	%	NO. OF CASES	%
< 2 HRS	0	0.00	0	0.00	0	0.00
2.00 -4 HRS	1	16.67	0	0.00	1	11.11
4.00 - 6.00 HRS	1	16.67	0	0.00	1	11.11
6.00 - 8.00 HRS	2	33.33	1	33.33	3	33.33
8.00 - 12.00 HRS	2	33.33	1	33.33	3	33.33
>12.00 HRS	0	0.00	1	33.33	1	11.11
TOTAL	6	100.00	3	100.00	9	100.00

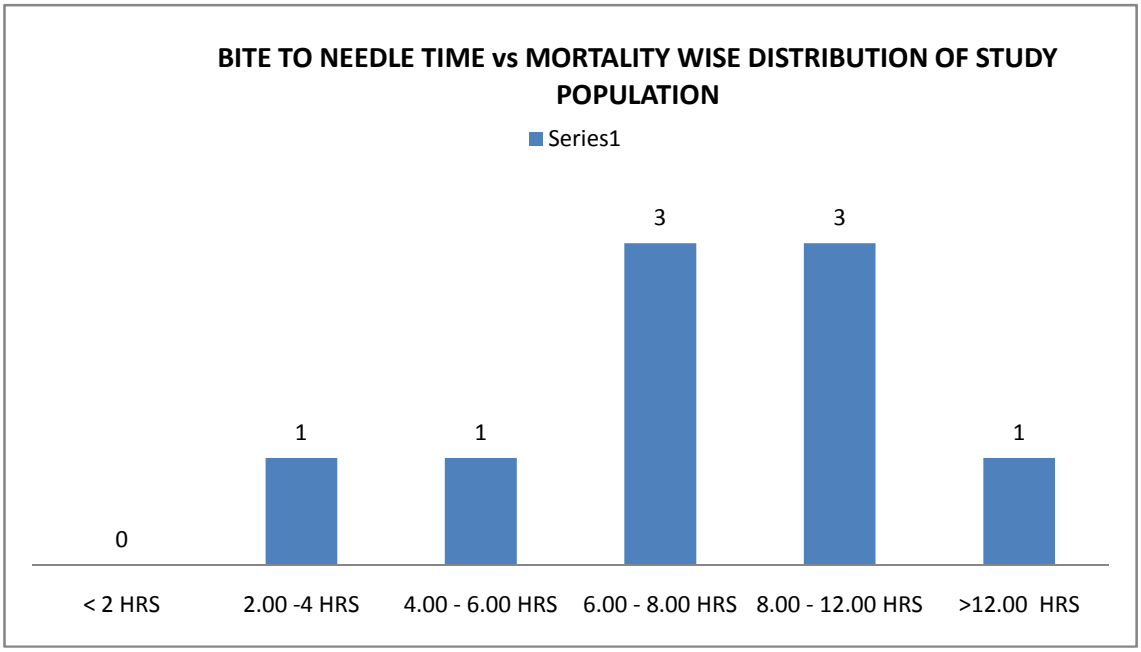
χ^2	0.415880232
As per table	11.07
Degree of freedom	5

$P<0.05$

So, the hypothesis is

accepted

FIGURE30: BITE TO NEEDLE TIME Vs MORTALITY WISE DISTRIBUTION OF STUDY POPULATION



Among 9 dead patients , 7 patients(78%) presented late with bite to needle time of > 6 hours and 2(22%) patients presented less than 6 hours from time of bite .

TABLE 15:BITE TO NEEDLE TIME Vs NIL MORTALITY WISE DISTRIBUTION OF STUDY POPULATION

BITE TO NEEDLE TIME	MALE		FEMALE		TOTAL	
	NO. OF CASES	%	NO. OF CASES	%	NO. OF CASES	%
< 2 HRS	18	10.47	6	200.00	24	13.95
2.00 -4 HRS	47	27.32	25	833.33	72	41.86
4.00 - 6.00 HRS	39	22.67	24	13.96	63	36.63
6.00 - 8.00 HRS	6	3.49	6	3.49	12	6.98
8.00 - 12.00 HRS	0	0.00	1	0.58	1	0.58
>12.00 HRS	0	0.00	0	0.00	0	0.00
TOTAL	110	63.95	62	36.05	172	100

χ^2 - 2.21983E-35 P<0.05

`FIGURE 31 :BITE TO NEEDLE TIME Vs NIL MORTALITY WISE DISTRIBUTION OF STUDY POPULATION

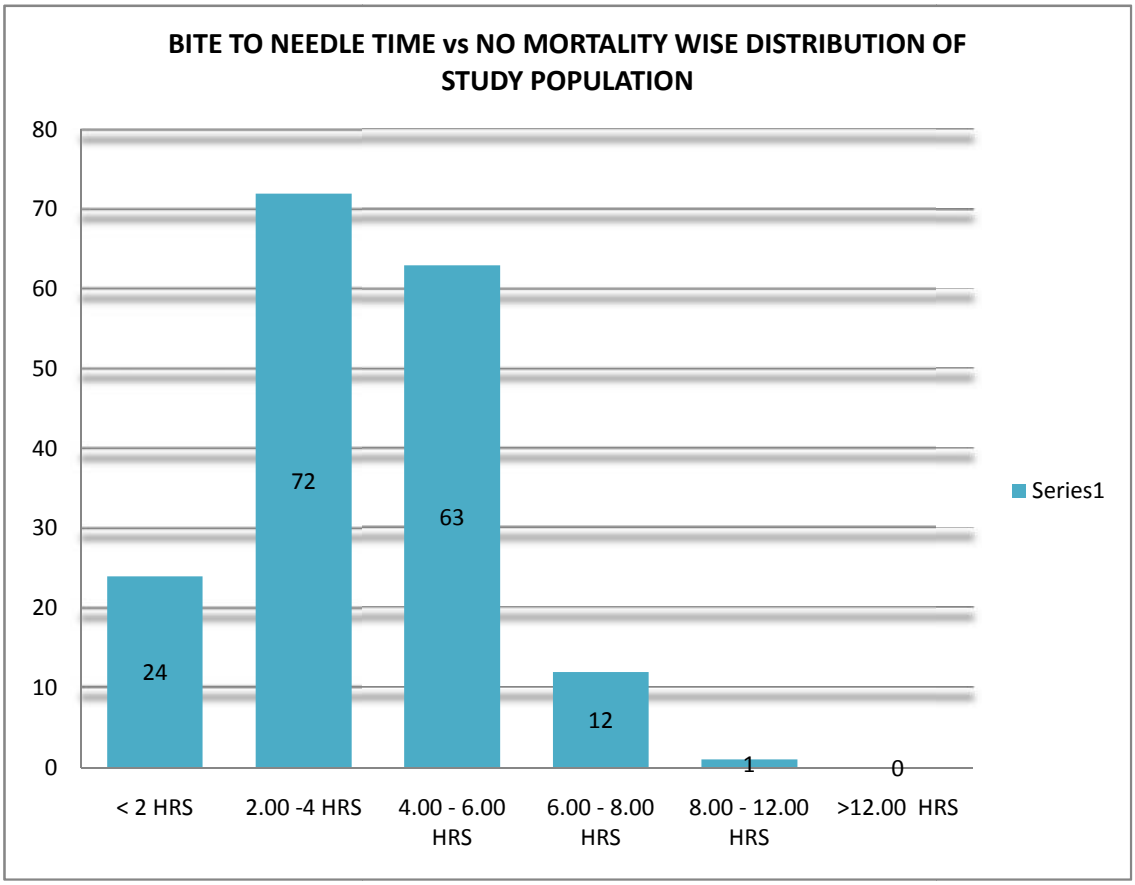
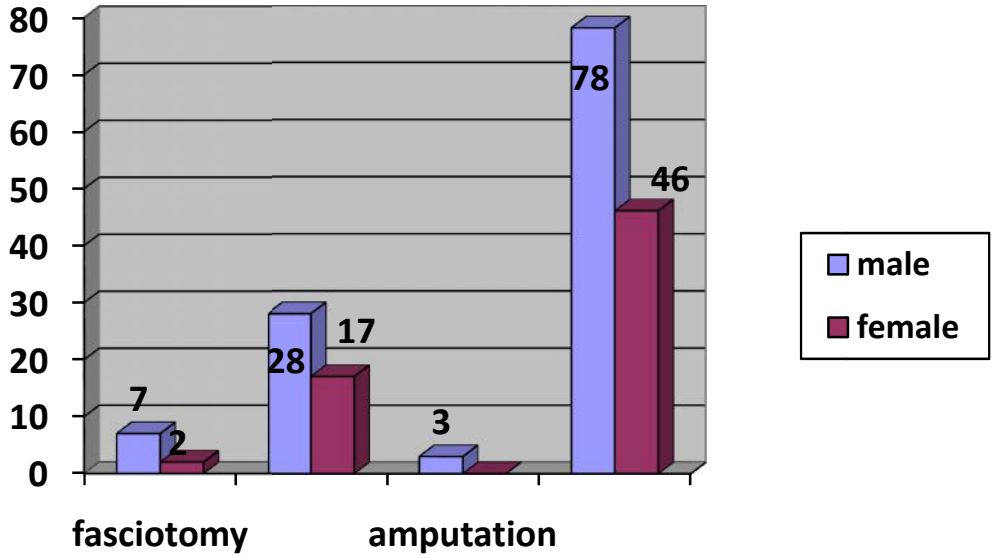


TABLE 16 :DISTRIBUTION OF PATIENTS BASED ON SURGICAL PROCEDURES

SURGICAL PROCEDURES	FEMALE	PERCENT	MALE	PERCENT	TOTAL	PERCENT
FASCIOTOMY	2	1.104	7	3.867	9	4.97
WOUND DEBRIDEMENT	17	9.392	28	15.47	45	24.86
AMPUTATION	0	0.00	3	1.657	3	1.66
NIL	46	25.41	78	43.-094	124	68.51
TOTAL	65	100	116	100	181	100

χ^2 - 1.20738E-40 P<0.05

FIGURE 32 :DISTRIBUTION OF PATIENTS BASED ON SURGICAL PROCEDURES



Of 181 patients 57 patients had undergone surgical procedures out of which 45 patients (24.8%) had simple wound debridement , 9 cases (4.97%) had fasiotomy and 3 patients (1.66%) had amputations . Remaining 124 cases (68.51%) got treated with out any surgical interventions .

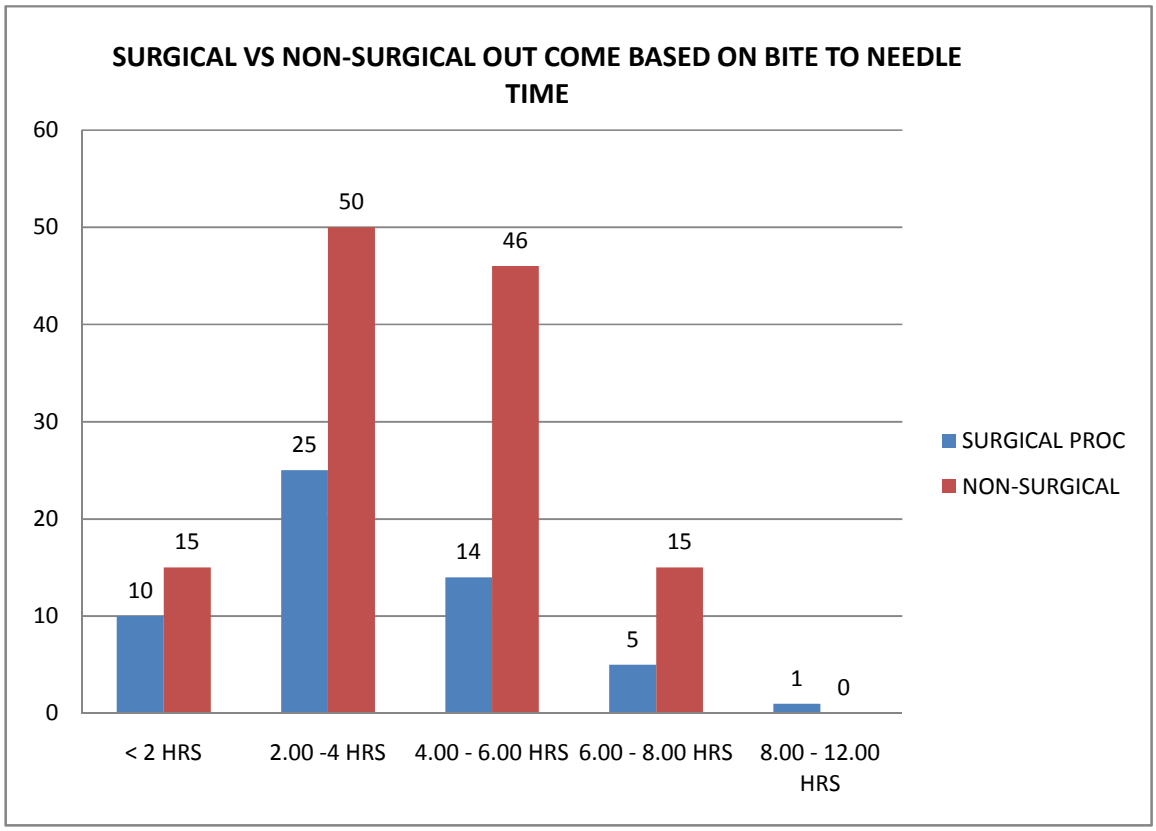
TABLE 17(a) :SURGICAL Vs NON-SURGICAL OUT COME BASED ON BITE TO NEEDLE TIME

BITTE TO NEEDLE TIME	FEMALE		MALE	
	SURGICAL PROC	NON-SURGICAL	SURGICAL PROC	NON-SURGICAL
< 2 HRS	2	5	8	10
2.00 -4 HRS	8	18	17	32
4.00 - 6.00 HRS	6	19	8	27
6.00 - 8.00 HRS	3	3	2	12
8.00 - 12.00 HRS	0	1	0	0
>12.00 HRS	0	0	0	0

TABLE 17(b) :SURGICAL VS NON-SURGICAL OUT COME BASED ON BITE TO NEEDLE TIME

BITTE TO NEEDLE TIME	SURGICAL PROC		NON-SURGICAL		TOTAL
	NO.	%	NO.	%	
< 2 HRS	10	40.00	15	60.00	25
2.00 -4 HRS	25	33.33	50	66.67	75
4.00 - 6.00 HRS	14	23.33	46	76.67	60
6.00 - 8.00 HRS	5	25.00	15	75.00	20
8.00 - 12.00 HRS	1	100.00	0	0.00	1

FIGURE 33:SURGICAL VS NON-SURGICAL OUT COME BASED ON BITE TO NEEDLE TIME



Out of 25 patients with BTN time < 2 hrs ,40 % had surgery and 60% without sugery. At BTN time of 2-4 hrs 33% with surgery and 67% without surgery.At 4-6 hrs values where 23% with surgery and 77% with out surgery. At bite to needle time >8 hrs 1 patient underwent surgery.

TABLE 18 :DISTRIBUTION OF STUDY POPULATION BASED ON TIME OF BITE VS SITE OF BITE

SITE OF BITE	4.00-7:59 AM		8.00-11:59 AM		12.01-3:59PM		4.00-7:59 PM		8.00 – 11.59 PM		12.00 – 4.00 AM		TOTAL	
	NO.	%	NO.	%	NO.	%	NO.	%	NO.	%	NO.	%	NO.	%
FACE	0	0.00	4	30.77	4	30.77	3	23.08	2	15.38	0	0.00	13	100.00
UPPER LIMB	6	13.33	3	6.67	14	31.11	6	13.33	5	11.11	11	24.44	45	100.00
LOWER LIMB	6	9.52	10	15.87	7	11.11	20	31.75	15	23.81	5	7.94	63	100.00
CHEST	2	7.41	7	25.93	17	62.96	1	3.70	0	0.00	0	0.00	27	100.00
ABDOMEN	0	0.00	3	14.29	6	28.57	1	4.76	10	47.62	1	4.76	21	100.00
BACK	0	0.00	6	50.00	5	41.67	0	0.00	1	8.33	0	0.00	12	100.00

FIGURE 34: DISTRIBUTION OF STUDY POPULATION BASED ON TIME OF BITE VS SITE OF BITE

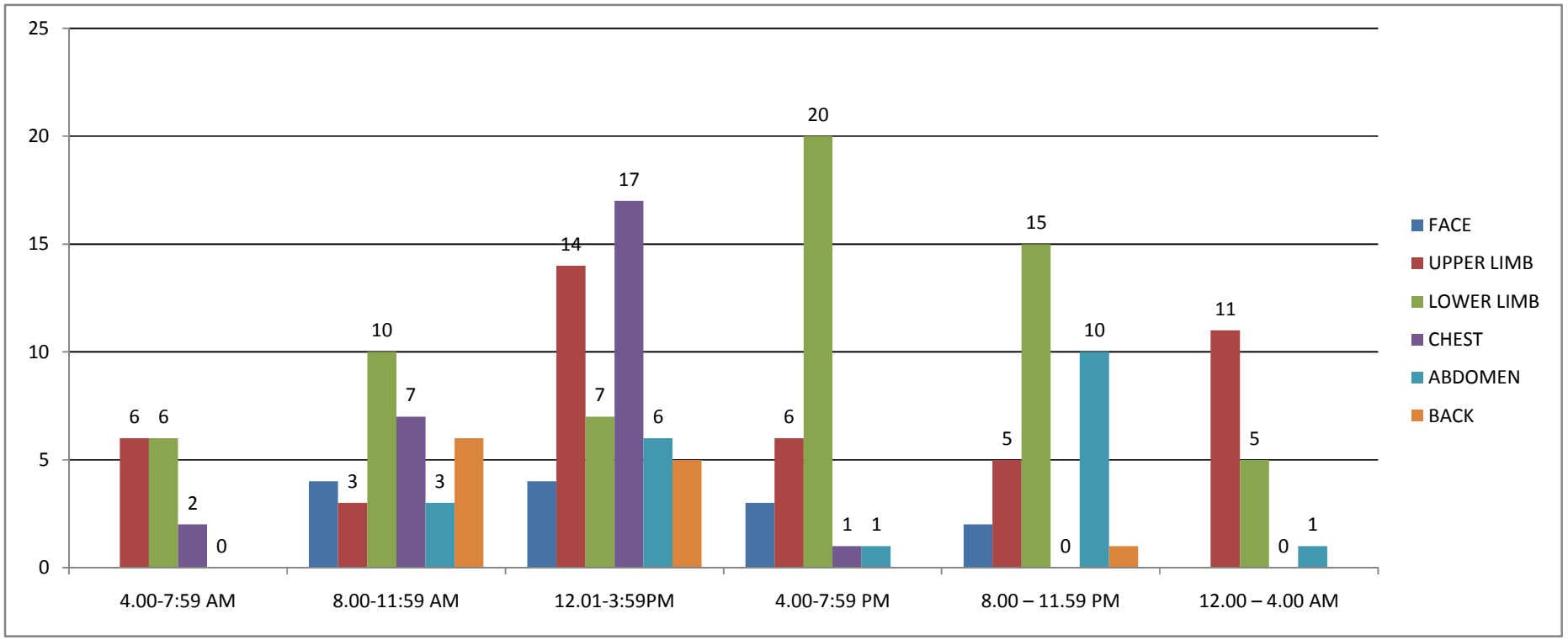


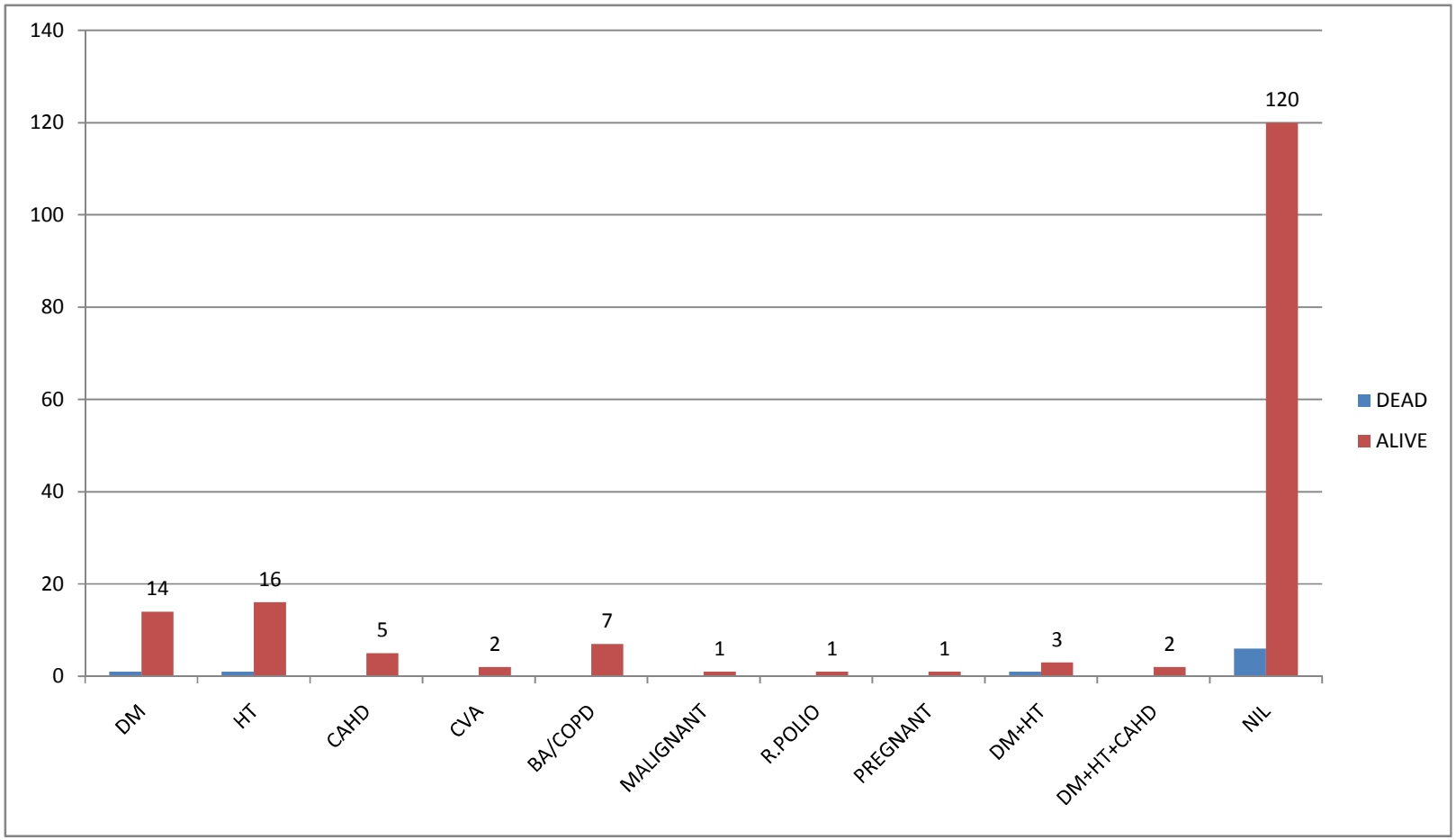
TABLE 19 COMORBID VERSUS DEATH WISE DISTRIBIUTION

CO MORBID ILLNESS	NUMBER	DEAD		ALIVE	
		NUMBER	%	NUMBER	%
DM	15	1	6.67	14	93.33
HT	17	1	5.88	16	94.12
CAHD	5	0	0	5	100
CVA	2	0	0	2	100
BA/COPD	7	0	0	7	100
MALIGNANCY	1	0	0	1	100
Residual polio	1	0	0	1	100
PREGNANT	1	0	0	1	100
DM + HT	4	1	25	3	75
DM + HT + CAHD	2	0	0	2	100
NIL	126	6	4.76	120	95.24
Total	181	9	4.97	172	95.03

χ^2	1.20738E-40
As per table	7.815
Degree of freedom	10

$P<0.05$

FIGURE 35:COMORBID VERSUS DEATH WISE DISTRIBIUTION



DISCUSSION

AGE:

Among 181 cases, most of the victims (26.8%) belongs to fourth decade of life. Many similar studies conducted in the past show similar distribution .One such study in JIPMER[60] show that majority of victims belong to 15-60 yrs of age. Another study by Sawai et al show that most of the cases belong to 10 to 30 years . There is arapid decline in the incidence of cases after 5th decade of life .

SEX :

In our study Male to female ratio of incidence of bite is 1.8:1 . Comparing this with studies done earlier like study conducted by Banerjee RN[61] ,a higher preponderance of males than females was observed .

OCCUPATION:

Similar to studies done at JIPMER[60] and in Safturjang hospital[61] our present study clearly showed that the incidence of bite is more among

agricultural workers i,e 46.41% in our present study .We also observed that 10 victims (5.52%) are school or college students .

IDENTIFICATION OF SNAKE:

In our present study we noticed majority of patients (60.77%) did not identify the snake . Among the identified cases , most common was cobra (16.57%) followed by krait (12.71%)

CO MORBID CONDITIONS:

To evaluate the outcome of patients in relation to co morbid illness , We obtained a deiailed history of co morbid illness among snake bite victims .Nearly 9.39 % of cases were hypertensives and 8.29% were diabetic . One patient was antenatal mother (5 months gestation).

TIME OF BITE :

In our present study ,maximum incidence of snake bite(29.28%) was between 4:00 PM to 8:00 PM . Least number of bites were distributed in morning hours i,e 4 to 8:00am(9.94%) and 8 to 11.59 am(9.94%).

SITE OF BITE:

Among 181 cases 50.83% of victims repoted to have bitten in lower limb and most common site was feet.other sites in decreasing order were upper limb (33.15%),abdomen (7.73%) and face (4.42%) .These findings clearly suggests that the site of bite is determined mainly by inadvertent contact of the snake during activities .All these findings are more or less similar to that of study conducted by Virumani SK[62] dutt OP and Bhat RN

PLACE OF BITE:

About 131(72 %)cases are outdoor (farm,work place, school ground,forest),

and 50(28%) cases are indoor bite (home, other buildings).

TOXICITY:

Neurotoxicity(35.38) is high among the toxicities observed . Next comes the pure haemo toxicity (25.42%) . Local reactions like cellulitis ,edema comprises 22.65 % .

BITE TO NEEDLE TIME:

Bite to needle time is the interval between time of bite and administration of ASV .42% cases were given ASV with in 2-4 hrs of bite , followed by 36% of cases with in 4-6 hrs . This helps in the evaluation of severity and administration of ASV in a crucial period

NATIVE TREATMENT:

In patients those who were alive ,history of tourniquet application was present in 48 (27.91%) cases, 7 cases (4.07%) had history of other methods of native treatment like cutting ,suction,etc. About 117 cases didnt recieve any native treatments . A significant association was found between tourniquet application ,other native treatments and study population. Out of 9 patients who were dead ,history of native treatment in the form tourniquet application, wound cutting, suction etc were present in all cases.

ASV REACTIONS:

Out of 181 cases 100 patients(55%) showed no reaction to ASV while in remaining 81 cases , 64 (35%) developed minor reactions in the form of

fever , urticarial rashes and rigors . 17 cases (10%) developed severe anaphylactic reactions. This clearly shows that majority of cases who received ASV developed no or only mild reaction to ASV .

BITE TO NEEDLE TIME AS A FACTOR FOR DETERMINING MORTALITY:

Among 9 dead patients , 7 patients(78%) presented late with bite to needle time of > 6 hours and 2(22%) patients presented less than 6 hours from time of bite.

SURGICAL PROCEDURES :

Of 181 patients 57 patients had undergone surgical procedures out of which 45 patients (24.8%) had simple wound debridement , 9 cases (4.97%) had fasciotomy and 3 patients (1.66%) had amputations . Remaining 124 cases (68.51%) got treated with out any surgical interventions .

COMORBID VERSUS DEATH WISE DISTRIBIUTION :

On analysing co morbid illness versus death wise distribution of study population , we found that among 15 diabetic patients 93.33 % were

alive and 6.67% were dead . Among 17 hypertensive patients only 5.88% died . Similarly on analysing all the co morbid datas we found that co morbid illness is less significant in determining the mortality .

ANTENATAL CASE OF SNAKE BITE:

We had one ante natal women (5 months of gestation) who received ASV in 4 hours and she was alive .But resulted in intra uterine death due to foeticidal effect of ASV . On analysing various ante natal snake bite cases in the past the foeticidal effect of ASV is confirmed .

SURGICAL VS NON-SURGICAL OUT COME BASED ON BITE TO NEEDLE (BTN)TIME :

On analysis of bite to needle time with surgery . we found that out of 25 patients with BTN time < 2 hrs ,40 % had surgery and 60% without surgery.

At BTN time of 2-4 hrs 33% with surgery and 67% without surgery. At 4-6 hrs values where 23% with surgery and 77% without surgery . At >8 hrs 1 patient with surgery . This shows that unlike bite to needle time as an important determinant of mortality , it's not an important determinant in case of surgical interventions.

CONCLUSION

Snakebite although a preventable problem , it remains to be one of the common emergency.

1) In the present study, the adult snakebite cases with envenomation brought to Government Chengalpattu Medical College & Hospital, Chengalpattu, were mostly males(64%)between the age group of 30 to 40 years(26.8%)

2)With rural background, snake bite is more common among agricultural related activities (46.41%)

3)The most common site of bite was lower limb and the maximum cases were recorded between 4:00 PM to 8:00 pm (29.28%)in the rainy season of July to September.

4) Neurotoxicity constitutes (35.38%) among all the toxicities followed by pure haemo toxicity (25.42%) and local reactions like cellulitis edema (22.65 %).

5) A significant association was noted between prolonged bite to needle time and mortality with 7 out of 9 patients(78%) who presented more than 6 hours after bite were dead .

6) On analysing co morbid illness versus death wise distribution of study population , we found that among 15 diabetic patients 93.33 % were

alive and 6.67% were dead . Among 17 hypertensive patients only 5.88% died . Similarly on analysing all the co morbid illness with outcome we found that co morbid illness is not a strong determinant in determining the mortality.

7)Native treatment especially tourniquett application is a strong determinant of outcome with 100% prevalence in dead and 85.28% in patients who had undergone surgeries and 27.28 % in alive patients with out surgeries .

SUGGESTIONS FOR PREVENTING MORTALITY :

Mortality due to snakebite poisoning can be reduced by taking certain steps:

1) Administer first aid as early as possible in case of snakebite poisoning

2) Always take the patients to the nearest health care facility for proper medical treatment

3) If the snake was identified ,should inform it without fail to the doctor to initiate early treatment based on expected toxicity.

4) Whole blood clotting time should be done in all the cases and ASV should be administered when it is prolonged.

5) Avoid native treatments like tourniquet applications, wound puncturing and suction. The bitten site should be immobilised and patient should be taken to nearest hospital

6) Always administer recommended dose of antisnake venom as soon as the patient is brought to the hospital , and do not wait for any pending investigations .

LIMITATIONS OF STUDY

1. Mortality is very low(9 out of 181 cases) to analyse the risk factor association
2. Smaller sample size
3. Chances of confounding bias are more
4. Absconded cases are excluded from study .

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PROFORMA

1.NAME /NO :	
2.AGE :	
3.SEX:	
4.OCCUPATION:	
5.COMORBIDS:	
6.TIME OF BITE:	
7.NO OF WARDS / IMCU STAY:	
8.SITE OF BITE:	
9.PLACE OF BITE :	
10.SNAKE IDENTIFIED :	
11.TORNIQUET APPLICATION	
12.SIGNS OF TOXICITY :	
13.BITE TO NEEDLE TIME :	
14.ASV DOSE AS PER NATIONAL PROTOCOL:	

15.REACTIONS TO ASV :	
16.BLEEDING TIME :	
17.CLOTTING TIME:	
18.RFT :	
19.PLATELET COUNT:	
20.CHEST X RAY:	
21.ECG:	
22.URINE R/E:	
23.OTHERS:	
24. HOSPITAL STAY > 5 DAYS	
25.SURGERIES :	
26.PERMANENT DISABILITIES :	
27.MORTALITY :	

KEY TO MASTER CHART

CODING

Q1. ENROLLED NUMBER OF THE PATIENT :

Q2.AGE : 1. 13 -18 YRS (MINOR) 6.42-47 11.72-77

2.19-24 YRS 7.48-53

3.25-30 YRS 8.54-59

4.31-35 YRS 9.60-65

5.36-41 YRS 10.66-71

Q3.SEX : 1.MALE 2.FEMALE

Q4.OCCUPATION : 1. AGRICULTURE 2.INDUSTRY 3.STATE GOVT (SWEEPER, SANITARY WORKER ,ETC) 4.CENTRAL GOVT 5.BUSINESS 6.SMALL TRADE 7.PRIVATE 8.NOT WORKING /HOME MAKE 9)FOREST WORKER 10)STUDENT 11)SNAKE HANDLER

Q5.CO MORBIDS : 1.DM 2.HT 3.CAHD 4.CVA 5.BA/COPD 6. MALIGNANCY 7. NEUROLOGICAL DEFICIT 8.NIL 9.CKD 10.PREGNANT

Q6.TIME OF BITE :1. 4-7:59 AM

2. 8-11:59 AM

3. 12-3:59PM

4. 4-7:59 PM

5. 8 – 11.59 PM

6.12 – 4 AM

Q7.NO OF WARD/IMCU STAY : 1.<24 HRS 2. 24 HRS TO 5 DAYS 3. 5-10 DAYS 4.10-15 DAYS 5.15-30 DAYS 6.> 1 MONTH

Q8.PLACE OF BITE : 1. FACE 2. UPPER LIMB 3. LOWER LIMB 4. CHEST 5. ABDOMEN 6. BACK

Q9. SITE OF BITE : 1. INDOOR 2. OUTDOOR

Q10.SNAKE IDENTIFIED : 1.COBRA 2. KRAIT 3.RUSELL VIPER 4.SAW SCALE VIPER 5. OTHER 6. NOT IDENTIFIED

Q.11.NATIVE TREATMENT : 1. TOURNIQUET 2.NO TOURNIQUET 3.CUTTING/PUNCTURING/BITING/NICKING

Q12.SIGNS OF TOXICITY : 1. NEUROTOXICITY 2. HAEMOTOXICITY 3.BOTH 4. LOCAL EDEMA / CELLULITIS 5.OTHERS

Q13.BITE TO NEEDLE TIME : 1. < 2 HRS/ 2.2-4 HRS/ 3. 4-6 HRS/ 4.6-8 HRS /5. 8-12 HRS/ 6. > 12 HRS

Q14.ASV DOSE AS PER NATIONAL PROTOCOL : 1. NEUROTOXIC (10+10) 2.HAEMOTOXIC 3.10 VIALS 4.8 VIALS

Q15.. REACTIONS TO ASV : 1. MILD 2.SEVERE 3.NIL

Q16. CLOTTING TIME : 1.NORMAL 2.PROLONGED

Q17. RFT: 1.NORMAL 2.ABNORMAL

Q18 PLATELET COUNT 1. NORMAL 2.ABNORMAL

Q19 CHEST XRAY : 1 .NORMAL 2. ABNORMAL

Q20 ECG : 1 .NORMAL 2. ABNORMAL

Q21.URINE R/E : 1 .NORMAL 2. ABNORMAL

Q22. STAY > 5 DAYS : 1 . YES 2. NO

Q23..SURGICAL PROCIDURES : 1. AMPUTATION 2.FASCIOTOMY 3.WOUND DEBRIDEMENT 4.NIL

Q24MORTALITY : 1 .YES 2. NO

Q25. VENTILATOR : 1.YES 2.NO

Q26.OTHER TREATMENTS : 1. FFP 2.DIALYSIS 3.NIL

q1 no	q2 ag	q3 sex	q4 occ	q5 morb	q6time	q7 stay	q8 place	q9 site	q10 id	q11 n.tre	q12toxi	q13 b to n	q14 dose	q15 react	q16 C.T	q17 RFT	q18 PLT	q19 CXR	q20 ECG	q21UR/E	q22 sta>5	q23surg	q24 p.dis	q25 mort	q26 venti	q27 othe
1	6	2	8	5	3	2	3	2	6	2	2	2	2	1	2	1	1	1	1	1	2	3	5	2	2	3
2	2	1	1	8	6	3	5	2	6	2	3	2	1	3	2	1	1	1	1	1	1	3	5	2	2	3
3	6	1	1	8	2	3	3	2	1	1	3, 4	3	1	3	2	1	1	1	1	1	1	1	5	2	2	3
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6	8	1	1	8	1	2	2	2	6	2	2	1	2	3	2	1	1	1	1	1	1	3	5	2	2	3
7	10	1	1	8	2	2	2	2	6	1	1	2	1	3	1	1	1	1	1	1	1	3	5	2	2	3
8	10	2	1	2	6	2	3	1	2	1	1	3	1	3	1	1	1	1	1	1	2	3	5	2	2	3
9	8	2	8	2	6	3	2	2	2	1	1	3	1	3	1	1	1	1	1	1	1	3	5	2	1	3
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11	1	1	10	8	1	3	2	2	6	1	1	2	1	3	1	1	1	1	1	1	1	3	5	2	1	3
12	7	1	1	3	4	2	3	2	6	2	2	2	2	3	2	1	1	1	1	1	2	3	5	2	2	3
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14	6	1	1	8	5	2	2	2	1	2	2	2	2	3	2	1	1	1	1	1	2	3	5	2	2	3
15	5	2	8	8	6	2	5	1	2	2	1	3	1	3	1	1	1	1	1	1	2	3	5	2	2	3
16	7	2	8	8	1	3	3	2	6	1	1	5	1	3	1	1	1	1	1	1	1	3	5	**1**	1	3
17	1	1	8	8	6	2	2	1	1	2	1	3	1	3	1	1	1	1	1	1	2	2	5	2	1	3
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21	2	1	1	8	1	2	3	2	6	2	1	2	1	3	1	1	1	1	1	1	2	2	5	2	2	3
22	5	2	8	8	1	2	3	2	1	1	4	1	3	3	1	1	1		1	1	2	2	5	2	2	3

23	2	1	6	8	4	2	3	2	6	2	3+4	1	1	1	2	1	1	1	1	1	2	2	5	2	2	3
24	5	1	1	8	2	2	3	2	1	2	5	3	1	3	1	1	1	1	1	1	2	2	5	2	1	3
25	5	2	8	8	5	2	3	2	6	2	2	1	2	3	2	1	1	1	1	1	2	2	5	2	2	3
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27	10	1	1	8	5	5	3	1	3	2	3+4	2	3	3	2	1	1	1	1	1	1	2	5	2	2	3
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32	4	2	6	8	2	4	3	2	1	1	3	1	3	1	1	1	1	1	1	1	1	3	5	2	2	3
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34	8	2	5	3	4	2	2	2	1	2	2	1	2	3	2	1	1	1	2	1	2	3	5	2	2	3
35	5	2	8	8	5	3	5	1	2	2	1	3	1	1	1	1	1	1	1	1	1	3	5	2	1	3
37	6	1	1	8	6	3	2	2	6	2	4	3	1	3	1	1	1	1	1	1	1	1	5	2	2	3
38	7	2	8	8	4	2	2	1	6	2	4	2	4	3	1	1	1	1	1	1	2	2	5	2	2	3
40	8	1	1	2	5	2	3	2	3	1	3	2	1	2	2	1	1	1	2	1	2	2	5	2	1	3
41	8	2	6	6	4	2	3	1	6	2	4	2	2	3	1	1	1	1	1	1	2	2	5	2	2	3
43	6	2	8	8	6	2	2	2	6	2	2	3	2	3	2	1	1	1	1	1	2	2	5	2	2	3
44	10	2	5	2	5	2	3	1	6	2	1, 4	2	1	3	1	1	1	1	1	1	2	2	5	2	1	3
45	7	1	1	8	5	1	3	2	1	2	4	1	4	1	1	1	1	1	1	1	2	2	5	2	2	3
46	1	1	10	8	4	4	3	2	6	1,3	3	3	1	3	2	2	2	1	1	2	1	3		1	1	2
47	11	1	1	5	5	2	5	1	1	2	1	1	1	1	1	1	1	1	1	1	2	3	5	2	1	3
48	4	1	6	8	2	2	3	2	6	1	4	3	2	1	1	1	1	1	1	1	2	3	5	2	2	3
49	7	2	8	8	6	2	2	1	3	1	4	4	2	3	1	1	1	1	1	1	1	1	5	2	2	3
50	4	2	2	8	4	1	3	2	6	2	4	2	2	3	1	1	1	1	1	1	2	3	5	2	2	3

51	8	1	5	2	1	2	2	1	2	2	1	2	1	1	1	1	1	1	1	1	2	3	5	2	2	3
52	3	1	8	8	5	2	3	2	6	3	4	2	4	2	1	1	1	1	1	1	2	3	5	2	2	3
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54	6	2	8	8	2	4	6	1	2	2	1	4	1	1	1	2	1	2	1	1	1	3	5	2	1	3
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